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OF THE

PHILADELPHIA NEUROLOGICAL SOCIETY.

VOLUME III.

1893-1894.

Reprinted from THE JOURNAL OF NERVOUS AND MENTAL DISEASE.

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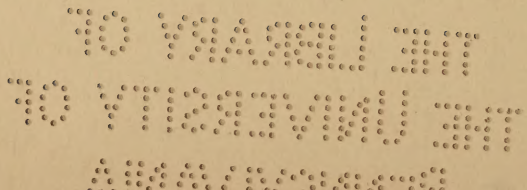

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PHILADELPHIA NEUROLOGICAL SOCIETY.

Stated Meeting, October 23, 1893.

The President, Dr. CHARLES K. MILLS, in the chair.

UNILATERAL SWEATING OF THE FACE AND NECK IN A PROBABLE CASE OF POSTERIOR SCLEROSIS OF SLOW DEVELOPMENT.

Dr. MILLS exhibited to the society this patient, E. R., 56 years old, white, who dates the beginning of his present troubles to an attack of grip three years ago, although this origin may be regarded as doubtful. He began to have what he termed rheumatic attacks in the legs; pains coming and going, but without any joint swellings. He does not describe his pains as having been shooting or lancinating, but rather as decided aches. Five or six times, however, during the last three years, he has had attacks of extremely sharp cutting pains across the left half of his chest, chiefly about the precordial region. These pains come in storms of agonizing severity, and he says that when the attacks are on he becomes delirious through his sufferings; as described, they correspond closely to the so-called "crises" of posterior sclerosis. In 1891, he was examined by Dr. John E. Carpenter, who again examined him a few days ago, and who reports that the same conditions are present now as then, namely, Argyle-Robertson pupillary reaction; constant myosis; poor vision brought up to normal by high astigmatic correction; ocular movements normal, and no signs of optic atrophy. Knee jerks are practically abolished, although a slight jerk can be brought out on the left with reinforcement; muscle jerk is present on the right, but absent on the left. His station is good and he has no ataxia of arms or legs. He presents the very curious feature of unilateral sweating on the left side of his face and neck. At times he also has a slight flushing or blushing of this side of the face. The case seems to point to an exceedingly prolonged first stage of posterior sclerosis. The unilateral sweating and blushing may be due to a patch

of sclerosis in the cervico-dorsal cord; or in the posterior ganglia, or perhaps in the left cervical gangliated cord. The case, in so far as the unilateral sweating and chest pains are concerned, recalls one presented to the society a few years since, which eventually proved to be thoracic aneurism; but, examination of the chest in this case is negative, and the combination of symptoms points to spinal degenerative disease.

Dr. MILLS also exhibited a case of

MIRROR-WRITING. (See page 12.)

DISCUSSION.

Dr. G. BETTON MASSEY.—A patient of mine, an artist, saw the programme of this meeting and asked what was meant by mirror-writing. I explained, and he remarked that he thought that he could do that. I have here a series of simultaneous both-hands writing which he did.

The first exhibit consists of words written simultaneously with each hand in the proper direction.

In the second, the right hand writes the word properly, while the left writes it reversed.

In the third, the right hand writes upside down and reversed, and the left hand upside down and backward.

In the fourth, one word is begun from the right and written upside down and another from the left upside down, backward and reversed.

Dr. F. X. DERCUM.—I find like Dr. Taylor, that I can without difficulty write in the reversed direction with the left hand, but if I attempt to produce mirror-writing with the right hand, it is accomplished with great difficulty. There seem to be physiological and anatomical reasons why mirror-writing should be adopted by children in whom the right brain has apparently assumed the principal functions of the left brain.

Dr. JAMES HENDRIE LLOYD.—I would suggest that the physiological action of the flexors and extensors of the arm probably has as much to do with the form of writing as anything else. In writing, the arm rests on the ulnar side. Hence the line or direction of least resistance, as it may be called, is away from the median plane of the body. So, too, the inclination of the letters will be fore-hand or back-hand according as the pen is held in the right or left hand. This is due to the nat-

ural action of the flexors and extensors of the hand when the arm rests on the ulnar side with the elbow-joint as a centre.

DR. S. SOLIS-COHEN.—In the Hebrew and some other ancient forms of writing, the characters are made from right to left. The reason probably is that the original crude-outline pictures were engraved on the rocks and onblocks of stone, the chisel being held with the left hand and the mallet with the right, and thus the natural method of progression was from the side on which the blow was struck, *i. e.*, right to left. When these characters were subsequently transferred to parchment or papyrus, the same direction was followed. If I am correct as to the reason for its original adoption, the fact, I think is in the line of Dr. Taylor's thought, that the mirror-writer goes from right to left, because that is the direction in which the left hand is most easily carried.

Dr. F. X. DERCUM.—We have here something more than writing from right to left. We have in addition a reversal of the form and position of the letters.

Dr. A. A. ESHNER.—I think that Dr. Mills in speaking of the natural tendency to centrifugal movement, at one point in his paper, brought out the explanation of this peculiarity of the hand-writing. The ordinary motions of the hands are made in the direction of least resistance, that is, those of the right towards the right, and those of the left towards the left. The phenomenon must be purely a motor one of the co-ordinative mechanism. In writing with the left hand it would seem easiest to transfer to that side the same sequence of stimulation and movement, as ordinarily occurs in using the right hand for the same purpose. That the peculiarity is essentially one of accommodation, would seem to receive support from the fact that, persons who write with the left hand and have their central hemispheres intact, slant the letters in the same direction as this boy does. In the latter, however, the governing mechanism is sufficiently well organized to overcome the natural tendency to compensatory aberration.

Dr. CHARLES K. MILLS.—I think that the suggestion of Dr. Taylor that any one can write in this manner, would, on experiment, be found to be incorrect. While it is true that Dr. Taylor can do this, we must remember that while not ambidextrous, he has a certain use of both sides of his brain which is not possessed by the average man. Dr. Taylor is also an artist. In my paper,

I have referred to the various explanations that have been given and also mention Hebrew writing. This is not the same as mirror-writing. Looking at Hebrew written in the mirror, would not give you the normal appearance of the letters. In the discussion too little stress has been laid upon the functioning of local areas of the brain itself, and upon the question of the formation of word images in the brain and the peculiar manner in which the consciousness of the image is registered upon the child's brain. This boy's left himisphere is probably out of use. It would be interesting to know exactly how the word image appears in his consciousness.

Dr. S. SOLIS-COHEN.—In discussing the question of natural writing we must consider the effect of imitation. If we were to take a world of left-handed people, probably all of the writing would be what we term mirror-writing. If then, a right handed man came among this people, his writing would be mirror-writing to them, unless he attempted to imitate their letters exactly, in which case his writing would have a backward slant. When a left-handed person comes into our world of right-handed people, he either writes mirror-fashion, as in the case cited by Dr. Mills, or he attempts to imitate the usual writing, and gives a back-handed slant to his letters. Coming back to those languages in which the writing is from right to left, of course the writing is not mirror-writing. It is not a reversal of itself. The question is, why does it take an awkward and apparently unnatural direction? The answer is that it was not originally unilateral dexter-handed writing upon parchment or other smooth surface, but a process of sculpture, requiring the use of two hands. The graving instrument being held in the left hand, the letters followed the natural line of the movement of that hand; the letters were thus cut from right to left and when imitated on parchment, were imitated in the same direction. It is significant in this connection that in such ancient writing there is no cursive hand. The letters are set down one after the other, by what is virtually a process of drawing and painting. It might not be impossible for right-handed persons to develop an alphabet or method of writing in which letters running into each other should require the hand to move from right to left, but it evidently was not readily developed as it does not exist. Again, in some of our present Roman characters, we

can trace mirror-pictures of the Semitic characters from which they have been derived. This seems to be because the right hand found it easier to make them reversed than like the original, just as most children and illiterate persons reverse the Roman S, making it Ꙅ.

Dr. WHARTON SINKLER.—It seems to me that there is something more than a mechanical difficulty in these cases. I recently saw a boy who is a little retarded in his cerebral development. He has been taught to read in words of three or four letters, but when he is tired, it frequently happens that he pronounces a word backwards, for instance the word "ton" may be pronounced "not," or the word "so," "os." That is evidently a defect in cerebration and not a mechanical difficulty. It seems to be an analagous condition to mirror-writing.

Dr. A. A. ESHNER.—In Dr. Sinkler's case, but a single visual impression was made upon the brain, and the direction in which it was viewed is of secondary significance, while in the case of Dr. Mills there are two distinct visual impressions. The boy sees the one picture and, through the motor mechanism, reproduces it in the simplest way.

Dr. J. MADISON TAYLOR.—It does not seem to me that mirror-writing can have a large significance, for writing with the left hand is not a difficult feat, provided that one writes in that fashion, viz., from right to left—reversing all the letters. I find that I can do this readily and rapidly, and do so on the blackboard before you. And further, the peculiarities of chirography are essentially my own. To write correctly from left to right is far from easy, however, and the result does not resemble my usual characters.

It would seem that to write at all with the left hand, to imitate the ordinary script of a right-handed person, one forms letters in a natural and easy *reversal* of the usual curves and lines pursued by the right hand, which have only become so by long pursuance of similar methods, and one of which comes the commonly accepted shapes and turns. And that as the left hand forms the curves there is merely a following along the lines of least resistance. For instance, it is a usual and an easy exploit to take two bits of chalk, one in each hand, and to draw a figure which has similar curves, as a butterfly with outstretched wings,—the two sides of the figure can be swiftly made by each hand at the same time.

Except for a lack of familiarity in holding a pen in my left hand, I can write very readily in imitation of the mirror-writing, and yet I trust no abnormality exists in my motor centres. On the contrary, I should infer, that my motor centre nerves were rather nimble to enable me to imitate my right-handed and accustomed methods by my left hand and reverse motor centres.

In the boy here exhibited, there may be right-handedness uncultivated, because his right arm has suffered damage; hence, he makes an unconscious selection in the easiest method of imitating this by his left hand, working at his greatest ease which is a reversal of direction and curves. He may be dimly aware of this, but his lack of intelligence prevents his being able to describe why he does so.

Dr. J. MADISON TAYLOR read a paper on

EXOPHTHALMIC GOITRE.

DISCUSSION.

Dr. G. BETTON MASSEY.—It seems to me that too much stress is laid by the reader of the paper, upon the thyroid portion of the disease, and also upon the positive relation of the sympathetic nervous system to the affection. The clinical studies that I have made, show me plainly that the sympathetic system has rather a negative relation to the disease. It is true, there is a relative over-action of the sympathetic system, or a loss of balance, but may not this loss of balance be due to a paretic neurosis of the spinal centre?—a mere prominence of the sympathetic vasomotor activity, by reason of its not being held properly under control by the opposing system. Hence, it is not a disease of the thyroid, and thyroid juice is hardly a method of curing so general a disease.

I agree with Dr. Taylor, that this is a far more curable disease than ordinary enlargement of the thyroid gland. I have seen several cases cured. Quite a notable one was sent to me in 1881, by Dr. Mitchell, for electrical treatment. The patient was a young lady with prominent eye-balls, a pulse of 140, and quite prominent thyroid. Under treatment continued for two years, she recovered not only from the affection of the heart, the pulse going down to 80 and losing its peculiar swelling character, but the eyes became normal in projection, and the gland very slightly above normal in size.

I think that the doctor, while enumerating many valuable means of treatment, dwelt too lightly on the use of the galvanic current. The use of the galvanic current presents possibilities of propriety and impropriety far greater than any other single therapeutic agent. We should not lightly say that a certain definite use has exhausted its possibilities. The case to which I have alluded was treated entirely by gentle galvanic currents applied both to the cervical region of the spinal cord and to the gland itself. In later cases, I have added to this, very heavy currents to the spinal region, with one pole at the pit of the stomach. I am quite convinced that the aberrant spinal impulses which fail to control in this disease, are directly stimulated by this treatment. I have seen the pulse go down immediately after the application. (Adjourned).

MIRROR-WRITING.[1]

By CHARLES K. MILLS, M D.,

Professor of Mental Diseases and of Medical Jurisprudence in the University of Pennsylvania, etc.

R——, thirteen years old, a strong, healthy infant, began to walk and talk at the age of ten months, and is reported to have had a sunstroke, followed by cerebral meningitis, when eighteen months old. Whatever the illness may have been it was attended with convulsions, and resulted in partial right hemiplegia.

The child's father was intemperate and dissolute.

The boy ranks high mentally, as compared with other children in the institution, and would be classed with the highest grade of imbeciles. He fairly understands all ordinary matters, as telling the time of day, the use of common appliances, the value of coins, notes, etc.; he can repeat the alphabet, can spell and read words of one syllable. He is a docile, sensitive, somewhat emotional child.

He was examined as to touch, pain, temperature, weight, resistance, pressure, etc., and no disorder of sensibility was found. Hearing, smell and taste are good.

The record of the examination of his eyes and sight as made by Dr. S. D. Risley is as follows:

O. D., V. equals $\frac{6}{\text{XXIVI}}$ O. S., V. equals $\frac{6}{\text{XXIV}}$

Vision is the same with both eyes together.

He matches colors perfectly. No contraction of the form field is present. At first in reading the letters on the test card he commenced to read them backward, but at the third line he read them as usual, that is, from left to right. The ophthalmoscope shows large central excavations in both nerves, and throughout the eye-ground the commencing pigments are absorptive, probably the result of eye strain. He has hypermetropia 2.D, with some astigmatism in both eyes. There are no eye-ground changes pointing to intracranial lesion. The lowered sharpness of vision is in part due to his refraction error.

---

[1] This boy is a pupil of the New Jersey Training School for Feeble-Minded Children at Vineland.

He has right-sided partial paralysis with atrophy. Movements about the shoulder and above the elbow are comparatively good, but as the arm is descended the loss of control becomes more and more marked, and is extreme for all the highly specialized actions of the forearm, hand and fingers. Practically all that he can do below the elbow is slightly to increase the flexion of the already partially flexed fingers, and then to extend these again to their usual positions. Strenuous efforts to move the fingers cause slight athetoid manifestations. Voluntary movements in the right lower extremity are affected much in the same way as the upper. The face on the right side shows a slight drooping, and a smoothing of contours; and he is unable to perform any special willed movements of this side of the face. The right upper extremity is much smaller than the left, and presents no contractures or deformities, except that the thumb is somewhat depressed and drawn under the index and middle fingers. The right leg, like the upper extremity, although considerably smaller than the left, is symmetrical, the only deformity being some adduction of the foot.

Some measurements were taken as follows:

	Right.	*Left.*	
Acromion process to external condyle of humerus. .	9.5	10	inches
Ext. condyle of humerus to lower end of radius. . . .	7.5	8.25	"
Dorsum of wrist to tip of middle finger.	6.25	6.5	"
Ant. superior spinous process of ilium to patella . .	13.5	14 5	"
Patella to malleolus.	13.25	14	"
Length of foot.	8.37	9.12	"
Circumference of middle arm.	7	8	"
" " forearm	8.75	7.75	"
" " middle thigh.	14	12.25	"
" " middle leg.	10.25	11	"

The photograph (see plate) shows the arrest of development of the limbs and body on the right side.

When efforts were made to teach the boy to write with his left hand from a copy it was discovered that he always made his attempts at writing from right to left, inverting the words and letters. A study of this peculiarity was then made.

Herewith is given a sample of his writing from a copy (see plate). He copies slowly, but always begins without hesitation to write in mirror fashion.

Mirror-writing is so called because it can be easily read in a mirror, in which the reflection appears as in ordinary writing. Erlemeyer, in a monograph published

in 1879, called particular attention to this curious defect, and it has received attention from a few investigators, particularly those interested in the study of brain physiology in its relations to disorders of speech and of writing, and to those who have discussed the dual action of the brain.

According to Savage,[2] mirror-writing is met with in some forms of mental weakness and in conditions of mental disorder allied to the hysterical; occurring also in cases of moral perversion where it may be only temporary; and being observed more commonly among women than among men, and being most easily acquired in highly nervous people.

In writing or copying a word from right to left, one would ordinarily trace the last letter and then the next, and so on backward to the beginning of the word. In mirror-writing the image of the word is inverted, the first letter in it appearing on the right and upside down, so that the word will be in the ordinary position when reflected from the mirror, or when seen from behind through transparent or translucent paper.

Mirror-writing can, of course, be done as a trick, or for amusement, and with the right or left hand, but those who try it will usually at first find it difficult to make particular letters, and more especially to connect them together consecutively.

Dr. Wilbur sent to Dr. Ireland specimens of the writing of a man who could write the same words with both hands at once, the writing done with the left being mirror-writing; but as he could also do the same with both hands moving from left to right in ordinary text, the performance was probably seight-of-hand.

Ireland[3] gives the details of several interesting cases, some of them much like our patient. One was a paralytic, imbecile girl, between eleven and twelve years old when studied; another was a genetous imbecile girl of fourteen; another, a congenital imbecile of twelve;

[2] Savage: Tuke's Dictionary Psychological Medicine, Vol. i., p. 573.
[3] Ireland: Brain, Vol. iv., October, 1881.

another, a boy of moderate intelligence of thirteen; all of these defective children wrote with their left hands the specimens of mirror-writing. He also mentions two left-handed idiot boys who formed pot-hooks from right to left, so that they would have eventually taught themselves mirror-writing. Buchwald, cited by Ireland, has reported the case of a right hemiplegic and aphasic, forty-five years old, who soon learned to write in a skillful manner, but in mirror-writing, his name as well as the numerals from one to ten, except the figure eight which he had forgotten. Although the inverse direction of his writing was pointed out to him, he could not be induced to try writing from left to right; and although by persistence he learned to copy some things correctly but awkwardly, he would again fall into the mirror-writing.

The left-handed show a physiological tendency to mirror-writing. Of a class of sixty boys and girls, who tried to write their names with their left hands, two girls and three boys wrote in mirror-writing, and all of these were found to be left-handed. Out of another set of one hundred and thirty-four children, six were left-handed, and three of these were mirror-writers. Imbecile children who are often left-handed also frequently show a tendency to reversions in spelling and in pronouncing words.

One of Leonardo da Vinci's manuscripts is an example of right-handed to left-handed or mirror-writing, and it has been supposed that this singular style was adopted to preserve the work from superficial readers, but another reason is suggested. A priest who visited Leonardo during the last years of his life, has recorded the fact that he had paralysis of the right hand, and it may, therefore, be that as he was unable to use his right hand, he learned to write with his left and became a mirror-writer.

A telegraph operator has informed me that at times when an operator is occupied in sending a message with the right hand, and wishes without stopping to make a

memorandum with the left, often the letters are reversed a B, for instance, being written ꟼ. Two explanations of this are suggested—one that the operator can with readiness write in the centrifugal direction from the trunk, and will, therefore, incline to reverse the letters; another, that the left hemisphere being intensely occupied with both the mental and manual effort required in sending a message, the right takes separate charge of the left hand in making the memorandum.

Ireland comments as follows on the probable mechanism of mirror-writing:

"It may be asked, is the image or impression, or change in the brain tissue from which the image is formed in the mind of the mirror-writer, reversed like the negative of a photograph; or if a double vision be formed in the visual centre, one in the right hemisphere of the brain and the other in the left, do the images lie to each other in opposite directions, *e. g.*, C on the right side and ↄ on the left side? We can thus conceive that the image on the left side of the brain being effaced through disease, the inverse image would remain in the right hemisphere, which would render the patient apt to trace letters from right to left, the execution of which would be rendered all the more natural from the greater facility of the left hand to work in a centrifugal direction. Moreover, when one used the left hand to write, there would probably be a tendency to copy the inverse impression or image on the right side of the brain."

Special convolutions in the right hemisphere have in a quiescent and undeveloped state the same functions which are active in the corresponding convolutions of the left hemisphere, as many observations on aphasic patients have demonstrated. Lesion of the left third frontal, or of the first and second temporal convolutions are restored or reacquired through the arousing and developing of the latent activities of the right hemisphere. The right side of the brain is not functionless, but its functions are in part, and sometimes largely, in abeyance. When the individual has the perfect use of the left half of his brain

—is right-handed, has the usual expertness with his right hand, has speech and vision in accordance with his inheritance and his training—impressions which come to him through his eyes are received and transmuted both by the lower and higher visual centres, so as to present to his consciousness a normal or a usual image, which, as a rule, has been registered only by the visual centre of the left hemisphere. In this centre the images are usually recognized as ride side up; the image formed on the right side, if one exists, is probably usually suppressed. When now the left side of the brain is destroyed, or when, as in the case of our patient, its development has been arrested, the individual is guided in writing by images formed in the right side of the brain.

Dr. S. D. Risley, of the consulting staff, whose examination of the boy is given in the report of the case, has kindly furnished the following additional notes:

"A little girl eight and a half years old, a bright, active, precocious child, in perfect health, had the right arm temporarily disabled from a fracture within the capsule. She was naturally ambidextrous. While unable to use the right hand and arm she went on with her rudimentary work at school, using the left hand for writing, making figures with as much apparent ease as the right had before been used, but the characters were formed from right to left and were waved as in typical mirror-writing.

"The second case was reported to me by Dr. John T. Carpenter, Jr. A gentleman, an expert stenographer, had his right hand shattered by a gun-shot wound and was compelled to resort to the use of the left hand, but found himself tapering into mirror-writing. When on his guard, *i. e.*, by intellectual attention, he could write correctly, but was much chagrined by often being compelled to trace what he had written by aid of a mirror. He was himself unable to read the page without such aid."

REPORT OF A CASE OF SPINAL ARTHROPATHY, IN WHICH MANY OF THE SYMPTOMS OF SERIOUS LESION OF THE CORD ARE ABSENT.

By WILLIAM J. TAYLOR, M.D.,

Professor of Orthopædic Surgery to the Philadelphia Polyclinic; Surgeon to St. Agnes Hospital; Assistant Surgeon to the Orthopædic Hospital and Infirmary for Nervous Diseases.

THIS case, lately under my care at the Philadelphia Orthopædic Hospital and Infirmary for Nervous Diseases, is of special interest, not only because spinal arthropathies are rare and our opportunity of observing them few, but because many of the more common symptoms of serious lesion of the spinal cord are absent.

The patient came to the hospital with the request that the knee joint be opened, the fluid which it contained evacuated, and asked that we operate at once.

Of the many physicians consulted by him, all pronounced the condition of chronic synovitis of the joint, and advised operation interference as the only means of relief.

Albert D. F., age 41, married and paper-hanger by trade, was first seen by me on August 15, 1893.

His family history is excellent.

He had the ordinary diseases of childhood, from which he recovered without incident.

When twenty-four years of age, he had inflammation of the lungs and later on, in the same year, an attack of erysipelas. In 1876, he suffered from an attack of inflammatory rheumatism.

All of the larger joints were implicated and he was confined to his bed for six weeks, but ultimately made a complete recovery. It is from this we must date the commencement of his present disability.

The following winter, or just sixteen years ago, he began to have pains in his limbs which he attributed to

"sciatica." These pains were similar in character to those from which he now suffers and have continued at intervals ever since. He states that no two attacks are alike in location, at one time the pain is in the toe or heel, at another in the calf of the leg, but most often in the anterior muscles of the thigh.

If he grasps the part suddenly at or above the seat of the pain, it will disappear in a few seconds. It will often re-appear in a few moments, but will invariably cease if the part is again grasped firmly with the hand.

These attacks of pain have continued with gradually increasing severity, until now they are well marked "lightening pains," and are always worse at night. There have been two attacks of localized ædema of the right leg, extending from the ankle to above the knee. The first five years, and the second two years ago. In each instance the swelling disappeared in a week's time, leaving the limb apparently in a normal condition.

The circulation in both limbs has been poor for the past five years, the feet and legs being cold both summer and winter.

He has always been of a nervous disposition and easily excited. He denies all venereal disease, and there is no evidence whatever to lead us to doubt his truthfulness.

He is a steady smoker of tobacco, but consumes only about four cigars a day. He smokes very slowly and these last him a much longer time than the average smoker. He uses a small amount of alcohol, but is not a drinking man.

In 1881, he had a small sore spot on the base of the right great toe caused by a nail in the sole of the shoe. This ulcer would not heal, and after it had troubled him for three months he consulted a physician who cauterized it with nitrate of silver. The ulcer then healed, but he states it had not been closed more than twenty-four hours when it became very painful. He then opened the wound with a knife and let out a quantity of fluid.

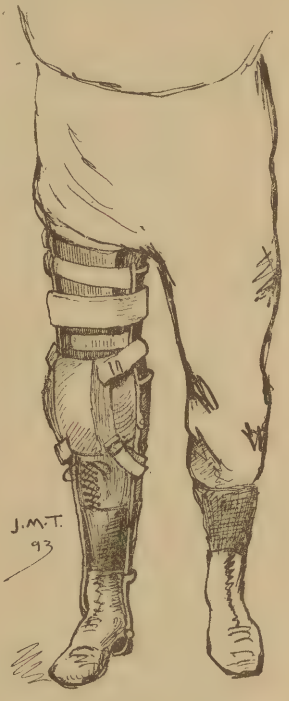
This state of affairs continued for about three years and gave him so much annoyance the toe was amputated at the mutatarso-phalangeal articulation.

The stump healed readily without further trouble.

His present condition began eighteen weeks before his admission into the hospital. He had been working hard for eight hours in a damp place and in a cramped position which brought great strain upon the right knee

joints. He states that during that day the knee felt heavy but gave him no pain, and had not pained him before this. At the close of the day he walked two miles to his home in a heavy rain storm and was wet through to the skin.

During this walk the knee began to pain and rapidly increased in severity until he thought he would never be able to reach home. The next morning the knee joint was much swollen, but gave him very little pain. From that day until now the joint has remained swollen and



enlarged, but has given him no pain whatever. The only sensation being one of weakness.

During the first twelve weeks, the relaxation of the joint increased, especially the ligaments on the outer side, producing a condition of outward bowing, but since this time the inner ligaments have given way and the joint is now markedly knock-kneed. Examination of the knee showed it to be much enlarged, the joint full of fluid which floats the patella.

The lateral ligaments are greatly relaxed and permit

of a range of outward and inward movement of nine inches. The knee is so relaxed that he cannot stand upon the leg and can only take a step with the aid of crutches. There is erosion of the head of the tibia and a separation of the epiphysis. There is marked crepitation when the bones are brought in contact, but there is no pain whatever.

There is a difference in circumference of the two knee joints of three and one-fourth inches.

His eyes were carefully examined by Dr. Thomson, and again three months later by Dr. de Schweinitz and Dr. Thomson, who made the following report:

"There is no ptosis, no stratisums and no myosis. The pupils are unequal and show the Argyll-Robinson sign, the dilated form, reacting readily to accommodation but not to light. The media is clear, the fundus is normal in O.D., and shows slight choroiditis in O.S., Both discs show the early stage of grey degeneration.

"Form fields are normal; the red and green fields contracted, the green proportionally more than the red. There has been no apparent change within three months, since the first examination."

All the other special senses are normal.

The station is good. There is no inco-ordination and no loss of power.

K. I., M. I. and E. I. normal.

Dynamometer, right 150, left 140; he is right handed.

Sensation appears to be normal, or possibly there is some retardation to pain and temperature sense in lower extremities, but this is not marked.

No loss of control over bladder or rectum.

The heart and lungs are healthy and the liver and splenic dulness normal.

Digestion is well performed.

He is of spare build, but states he has lost weight during the past eighteen weeks.

He states he has never noticed any change in his sexual appetite or power, neither an increase or diminution.

I believe conservative methods of treatment in these arthropathies of spinal origin should always be preferred.

A number of cases have been operated upon by different surgeons, and attempts at resection of the joint have nearly always resulted in failure.

The trophic changes which permit of the destruction of the articulation act also in preventing union of the wound surfaces.

Some years ago it was my misfortune to have under my care a slight wound of the sole of the foot in an ataxic. The wound itself was slight and simply through the skin, but my patient nearly lost his life, and recovered only after many weeks of great suffering.

Here the destruction of the joint is so great, the separation of the epiplysis of the tibia, the enormous enlargement of the serus surfaces and the dislocation backward of the tibia upon the femur so marked that I do not think it would be possible, disregarding the trophic cause, to expect anything other than failure.

An amputation in the upper one-third of the thigh might possibly result in union of the stump, but this I doubt.

He was kept in bed for some time at complete rest; and with the rest the swelling of the knee diminished somewhat. He was suspended daily an increasing number of minutes with the happiest results in relieving the pains. Potassium iodide was given internally for a time.

In the hopes of relieving pressure and jar to the knee as well as support to the relaxed joint, I had made for him a brace. This brace consisted of two stout bars of steel extending from the upper portion of the thighs to the sole of the foot, with joints at the ankle and knee, with several semi-circular bands of steel for strength. This was attached to a shoe by the ordinary stirrup.

At the knee is a lock to keep the leg extended and yet permit of bending the knee when he is sitting down.

The portion which surrounds the thigh is made to resemble somewhat the socket in an artificial leg for a thigh amputation. This completely encircles the thigh, is strapped tightly to it, and thus relieves pressure upon the knee joint by supporting the weight of the trunk.

A broad band of webbing is strapped over the swollen joint to give it fixation and pressure. The accompany-

ing photograph gives a much better idea of this apparatus than my description of it.

Of course, no claim is made that this will in any way cure the condition, but it has been of the greatest service to him.

When he first came to the hospital, he was totally disabled, and could stand only with the aid of crutches.

Now, for nearly three months, he has been able to walk around with comfort and without the aid of a cane. He tells me he has been at work, has papered quite a number of houses, and although the stiff knee makes it awkward for him at times he has no pain.

Of course, he cannot walk as far or for as long a time as before the knee joint was affected, but he is able to stand and walk with ease and comparatively little discomfort.

For the very careful notes of this case, I am indebted to Dr. Walker, the resident surgeon at the hospital, and for the photographs to Dr. Boyce, also of the house staff.

These latter show most accurately the appearance of the enormously enlarged knee joints with the relaxation of the ligaments and the brace when applied to the limb.

A CASE OF MULTIPLE NEURITIS, SIMULATING LANDRY'S PARALYSIS IN THE RAPIDITY, ORDER AND EXTENT OF PARALYSIS.

By F. SAVARY PEARCE, M. D.,

Philadelphia.

THROUGH the invitation of Dr. J. Madison Taylor, and by the kindness of Dr. S. Weir Mitchell, I report the following case. The simulation of the multiple neuritis in this man to the so called paralysis ascendens acuta (a disease rare and whose course and clinical features are not described alike by many authors) was a somewhat confusing element in the differential diagnosis, (if, indeed, any exist) at the time of admission of the patient to the Infirmary for Nervous Diseases on April 3d, 1893.

A. B., male, æt. 59 years. In the family history it is noted the relations were as a rule healthy and long lived. Two brothers had died in infancy, one as the result of an accident. A third brother succumbed to some bowel trouble at 27 years of age. Five sisters are all healthy at middle life. Father had died of old age; mother of apoplexy, æt. 68. No other neuroses or psychoses and no alcoholism.

The patient was always a healthy boy, had the ordinary diseases of childhood in mild forms. Boasted of his strength as a young man. Has never married and denies alcoholism or venereal disease. Thirteen years ago had inflammation of bowels.

Was wounded during service in the Civil War near outer malleolus of right leg and foot by a "spent ball," which was removed at the time, but the ankle bones were so crushed as to lay him up practically for two years. There was apparently no neuritis following the injury, but he has been lame ever since. Has never been able to do much manual labor since, and has not done any work for the past eight years. This excludes toxication (from the occupations' usual sources of poisoning) at

least in the ætiology of the present trouble, which dates back seven weeks previous to admission to the hospital. The patient had been in good health and walking about up to February 16th, 1893. The next day he complained some of nausea, of "belching of wind," but no vomiting and no diarrhœa. Had a sense of malaise; later in the day felt weak in his legs and on waking February 18th, it was noticed he had some difficulty in articulation. His tongue felt thick. The legs, hands and arms had now become feeble. Patient walked about the house some, but later in the day the nausea recurring, a physician was summoned who pronounced the trouble "acute indigestion." The sister says he now had some fever. No flux or abdominal tenderness existed then, nor since. On arising Sunday, February 19th, (about 48 hours after the first attack of nausea) patient felt very weak. Said his knees felt as though they would give way, and when he got down stairs fell from inability to bear up the body weight by his enfeebled extremities. On attempting to walk again he fell a second time, after which he was assisted to the breakfast table where he ate a hearty meal, but by noon he became extremely weak all over his body, so much so that he could not feed himself and was carried to his room that night having considerably fever. There was no acute pain at any time and it did not cause pain to be lifted.

There seems to have been no tenderness along the nerve trunks even from the first. He now remained in bed, the helplessness increasing rapidly in the inability to move legs, then arms and trunk, (although the power of aaticulation soon returned) until at the end of about 36 hours from the onset of this fulminating course of the paralysis he became almost completely diplegic. The fever gradually subsided, and his bowels moved normally and regularly as they had not done previous to present trouble.

He remained in this helpless condition and three weeks later fell from a chair he had been lifted into, (this was four weeks before admission to hospital) bruising the dorsi of both feet, it was thought. At any rate symmetrical greenish ecchymoses are found here at the first examination. There is also slight œdema of feet. He has no trouble with the kidneys, bladder or bowels, before or since the paralysis.

There has been no loss of sensation and there are no evidences of trophic change any place on the body.

Patient has had erections normally up to within a week of admission. The voice has gradually returned to normal, the only difficulty now being slight dypnœa and a tickling sensation in the throat causing hacking and expectoration of small amounts of mucus.

On admission too, the lungs are clear, (respiratory sounds faint) the temperature is 101°. Pulse, rapid and weak. No cardiac murmur. Is of fairly good physique. Liver and spleen of normal size and not tender in palpitation. The man's teeth are bad. No blue line. The bowels continue regular. There is slight tympanitis from abdominal paresis. Tongue clean. Appetite fairly good. Hearing good. Very soon the slight nervous cough lessened, and while he had no subjective pain, yet he bore an anxious expression, as one suffering from some very acute disease. He is entirely helpless. There is no face or tongue palsy, however, and the latter is protruded straight. Takes semi-liquids readily as fed by the nurse and there is no regurgitation. Can shrug his shoulders slightly. Can move toes and fingers a very little. Can flex knees slightly when supported on the bed. Makes slight quivering movement of abductors of thighs, not so of adductors. The muscles of the thighs, legs and arms, are "mushy" to the touch. The right thigh and leg are somewhat less in size than the left, due to old injury to foot and leg. The great toe of right foot is in position of hallux valgus, also probably induced by the gun shot wound of foot.

The sensation in both lower and upper extremities is unaltered as to touch, temperature or pain, excepting of a slightly tender point on deep pressure in the left popliteal space; none along other nerve trunks. There is some muscular soreness in the palms of hands and soles of feet, rather than a true hyperasthesia. The joints of fingers on both hands are sore on pressure, not to light touch. Not so in the toes. Tells pin prick any place readily. Tells point from head and there is no delay. Knee jerk is absent and not reinforcible by making a grimace, the only voluntary movement of any movement remaining. Plantar, cremasteric and abdominal reflexes nil. Dynamometer, r. o., l. o.. Jaw jerk is good, being the only reflex not affected, excepting the others of the head. The muscles react very slightly to blow of percussion hammer, especially so in flexors of forearms. After blow, muscles maintain a tetanic contraction, remaining "hard" a few seconds to half a

minute. This is seen even in as small muscles of the hands as the first dorsal interossei, and more on right than on left side. The pectorals hump in like manner. Sphincters are normal. He sweats a great deal and the hands have "sour bread" smell, yet have been washed frequently.

The man's breathing is curious. There is little, if any movement of abdominal muscles or viscera. The respirations are feeble, hurried and seem to be costo-diaphramatic in type, the latter muscle not working satisfactorily, but producing jerky breathing.

Electrical examination by Dr. I. P. Willitts, was reported as follows, April 7th, four days after admission:

Faradic.—Two and a half inch coil, secondary; produces contraction in extensors both legs, most marked in tibialis anticus; same in arm extensors, flexors same.

Galvanic.—Five m. a. gives An Cl C = K Cl C in extensors of left leg; Seven m. a. gives K Cl C > An Cl C in extensors right leg. This shows marked quantitative change in all muscles affected with possible De R. commencing in left leg. This in seven and a half weeks.

Dr. De Schwainitz examined the eyes and reports, "with exception of sleight œdema of nasal edges of optic discs, there are no changes in the fundi. Vessels and form fields normal. Pupils respond to light and accommodation, but the left is a little the larger."

Thus, four days after admission and after this study of the case, (the crisis apparently over) Dr. Mitchell ordered continuance of diet confined to milk and vegetables largely, quinine sulph. gr. xii daily. Bowels to be kept open, and in active therapeusis alternating hot and cold baths to arms and legs b. d. There was absolutely no disturbance of his digestion as we found up to a week after admission and his diet was increased accordingly. Urinalysis negative. At this time also the temperature ranged from 100° to 101°. The paralysis which had seemed about to affect the medulla so seriously had not progressed. Examination of the spleen still revealed no enlargement or tenderness. The greenish spots of ecchymosis persisted over most of the dorsi of feet. No pain on pressure along nerve trunks. The only subjective symptom is a sense of formication in the extremities much relieved by the hot and cold baths. Muscular reactions to-day, (April 10th):—chest = normal reactions and humping on tapping with percussion

hammer. Left arm: deltoid humping marked. Excessive humping in biceps both sides, the primary humping being feeblest. There is scarcely any reaction remaining in flexors of forearms. Both primary and secondary reactions very good in extensors of forearms, the humping continuing for a long time after the blow is struck. Also, he can shrug shoulders a little better. Can contract adductors of both thighs some but not enough to move limbs, The little gain in flexion and extension of fingers is less marked in left hand. Abductor muscles have gained some in power, but he can not move limbs out as he lies utterly helpless upon his back.

April 11th.—His temperature is 101°. Sleeps well, appetite good. The cough has ceased and lungs are clear. The plantar, knee, elbow, abdominal and cremasteric reflexes are still absent. There is faint fibrillary contraction on tapping anterior tibial groups and gastrocnemii both sides.

April 12th.—Patient had a good night's sleep. Today the slight movement of fingers is better in right hand, the first dorsal interossei muscle being the most active muscle on either side. Tapping this muscle produces marked fibrillary contraction. He has also recovered the use of abductors of thighs sufficiently to move limbs out a little as he lies on back. Takes nourishment well as fed to him.

April 13th. — Only change noted was that the fibrillary contraction did not exist while longitudinal contraction could be elicited a little in the anterior tibials. Longitudinal contraction was pretty well marked on tapping the gastrocnemii; a persistent fibrillary quivering continuing for some time after. The reaction to stimuli of both recti is fairly good. There is a slight plantar response easily exhausted on left, none on right side. The next day the muscles of arms were in better tone, excepting those of forearms. The right plantar reflex has returned feebly but the left could not be elicited. This vacillation of plantar reflex activity has been very marked. K. J. is absent. No tendo-Achillis reflex. The patient weighs 130 lbs., not much below his usual weight.

April 15th.—Dr. Kyle made cultures from blood taken from extravasations on dorsi of both feet, but with staining and high power no micro-organisms could be found. Thus the cause of the paralysis, if due to bacteria, was beyond our reach. That some infectious process was

going on (possibly aptomanic poisoning) seemed inevitable, a blood count showing, out of 4,000,000 r. b. c. to c. m., only about 3,000,000 healthy cells. The remainder of the red corpuscles were irregular in form, crenated and many contained granular matter. Hæmoglobin estimate on admission being 68%, now was reduced to 50%. The white corpuscles were slightly increased. Surely all these were evidences of a profound dyscrasiac!

By the 18th of April, fibrillary twitching was everywhere lessened, and on the 20th, even more so, when too, the left plantar reflex had again reappeared. Measurements show right leg to be the smaller one, due to old injury stated, and not to unilateral acute wasting. But little difference existed in the thigh.

Massage and faradism was begun on the 23d of April, (10 weeks after beginning trouble) producing soothing and agreeable effect. He had no subjective pain. On the 19th an electrical examination had proved well marked reaction of degeneration existing in both legs, worse on right.

Galvanism: Left leg extensors, 6 m. a. An Cl C > K Cl C.

Galvanism: Right leg extensors, 8 m. a. An Cl C > K Cl C.

April 27th.—Sensation to heat, cold and touch perfect. Freezing part by ice and rhigolene had no effect on reflexes. The K. J. and elbow jerk remaining absent, the jaw jerk was well marked. On the first of May it was noted:— the right leg moves slightly more, and the right plantar reflex is increased in force. He breathes more by diaphragm, respirations less jerky, and liver now rises and falls with respiratory movements.

The same measurements of lower extremities obtained on May 8th as had been recorded April 20th, 1893.

May 20th.—All fibrillary contraction has disappeared. Cremasteric reflex has returned slightly.

Thinking of possible poison from arsenic in wall paper or from other source, a specimen of the man's urine was sent to Dr. John Marshall, at the University, who reported negatively. By the first of June the extension of paralysis was stayed, and he has made very slow improvement since as regards motion. He can now flex and extend right leg, adduct well, abduct slightly. The left leg can be adducted to lift foot 12 inches from bed, the patient being held on left side. Sensation seems perfect. If placed on right side can turn on to back.

Can shrug shoulders well. Can pronate, adduct and but slightly extend arm at elbow on both sides. Perfect control over bladder and bowels. Temp. 100°.

June 17th. — The note was made: No subjective pain anywhere. None on pressure over nerve trunks. No bad sores. Can adduct, abduct, supinate and pronate arm by shoulder muscles. Holds pencil in fingers for first time. Elbow jerks absent. Arm muscles respond faintly, left side best. Plantar reflex improved. The muscle jerks in legs are better on the right than on left side.

July 31st.—Faradic current gives no response in extensors both forearms. Three inch coil contracts extensors both legs.

Galvanic current: An Cl C > K Cl C in both forearm extensors = De R. present. This shows gain below and loss in upper extremities.

August 18th. — Slow gain. Diaphragm used more. Sensation to touch, temperature and pain normal. All motions improved. Joints of fingers are stiff from disuse. No K. J. or elbow jerk. Attempt at clonus in right foot. Leg muscle jerks good. Cremasteric and plantar reflexes normal. Temperature at night 100_0, pulse 93° to 95°. By September 10th there was no decided change. The movements were slightly improved. Was now put on strychnia gr 1.40 t. d.

October 25th. — Sensation good. Motion much improved in hands and arms. Can touch nose with hand for the first time. No elbow or muscle jerks in arms. No K. J. or clonus. Plantar and cremasteric reflexes present. Muscle jerks in legs present. Can roll over on side in bed. Chest expands better in breathing. Controls bladder and bowels perfectly. Has erections normally.

November 1st.—Electrical examination by Dr. Willets:

Faradism.—Lost in all extensors forearms and legs, and in triceps.

Galvanism.—12 m. a. is necessary and gives An Cl C > K Cl C. in all the affected muscles. De R. present.

November 14th.—Patient's expression duller. Temp. 101°. Put on Ext. Thyroid gr. x. t. d., on empty stomach.

November 20th. — No change except that the temperature which has been running up to 99° and 100° has not been above 99°, and pulse is slightly more rapid. Reflexes same as last note. Can move all parts better.

November 23d.—Some dizziness when turning in bed. None when up on chair. Temperature not above 99° since taking thyroid. Pulse more rapid (increased from 110 to 120). Has same trophic change about great toe nail of left foot. No bad sores. Blood count is over 5,000,000 red blood cells, 75% hæmoglobin.

Status Præseus. — December 26th.—Patient was too helpless to be brought before you to-night. All muscles of upper and lower extremities are wasted and flabby. Interossei muscles, both hands, are very much wasted, making the hands passively claw-like. Can slightly flex, extend, adduct and abduct fingers, both hands, but the movements are clumsy. Complete wrist drop, both sides and on supinating forearms for him, he can not flex wrist, which can be done slightly, however, when not so impeded by gravity. Can put hands on head by action of shoulder muscles mainly, the forearms being flail-like. Muscles respond feebly to stimuli. Chest muscles are also flabby and pectorials act feebly. Jaw jerk, good response. K. J. and elbow jerk absent. Cremasteric and abdominal reflexes faint.

Can raise both legs high off bed; but the effort soon tires him, and the members drop flail-like on the mattress. Can adduct and abduct fairly well. Can flex and extend ankle and toes slightly. There is the characteristic ankle drop.

No tenderness along nerve trunks. No bad sores. The nails of first and third toes of left foot are now undergoing trophic change. All functions and emunctories fairly normal. Bowels moved daily. Urine also voided normally. Heart's action is weak. Chest expansion is poor.

To sum up—There first appeared nausea, difficulty of speech and of respirations. Then there was general weakness; finally sweeping like a cyclone the whole voluntary motor system below the head, beginning in lower extremities, extending rapidly to trunk and arms. These were preceded by malaise, fever, formications, but accompanied by no pain or tenderness along nerve trunks. All this took place too, within two or three days at least, placing a man in full vigor apparently, upon his back in utter helplessness, and from the history what would appear to have been grave bullar disturbance, the latter soon disappearing, although the vagi were weakened in their control over diaphragm, and the external respiratory nerves lost power over the serrati for weeks after the crisis was over. The adductors of thighs were more and sooner affected than the abduc-

tors, especially noted at end of first week after admission. The extensors or anterior tibial group were affected before (and more profoundly ever since) than were the posterior muscles of the legs. The extensors of forearms wasted before the flexors.

All these, with the rapid loss of reflexes, no early trophic changes, no history of alcoholism or other exciting cause, were a group of symptoms much resembling those of acute ascending spinal paralysis, so called. The age of the patient, the electrical inaction of the muscles; more in legs at first, then in arms, and, in July, '93, the gain (since April) in electrical response, of lower extremity muscles (even from the apparent degeneration of anterior tibial muscle of left leg) over those of the upper members, where degeneration had then taken place; and finally, the degeneration found in November, '93, of all affected nerve supply, (confirming the order of paralysis given above) are of course, *prima facie* evidence of the neuritis from the first and now existing.

Prognosis is unfavorable.—The increase in control and range of motion in degenerated states of nervus supplying the parts is always interesting, probably showing specialization of the remaining little or unaffecting fibres.

Strumpell says,[1] "there is usually no rise of temperature in ascending spinal paralysis." He thinks the symptoms must be due chiefly to functional disturbance from some toxic influence, and further states that "the condition of the reflexes and the rapid loss of electrical excitability in connection with the pains at the beginning seem to justify the hypothesis that the disturbance sometimes has its chief seat in the peripheral motor nervus, the disease then exhibiting the most acute form of infectious multiple neuritis," and finishes the pathogenesis as follows: "In other cases, however, the motor portions of the spinal cord, the lateral columns and the anterior gray cornua are, perhaps, chiefly affected." If there is recovery it is usually in a few weeks. Wood says,[2] "weakness, irregular formications, spots of numbness, weariness and discomfort," etc., are the prodromata of Landry's Paralysis. That paralysis begins in foot muscles, then legs, thighs, arms and in muscles of respiration. That the temperature is rarely above normal, and there is no pain (only discomfort, formications or slight numbness). No trophic changes. Sometimes marked anæs-

[1] A Text Book of Medicine, p. 637.
[2] Nervous Diseases and Their Diagnosis, p, 51.

thesia. Usually none. "Œdema of skin" and marked sweating (as in this man) exist in some cases.

In Ross's thorough work[3] published this year in connection, Bury, most of the reported cases of Landry's paralysis are tabulated. He records 93 cases, and throwing three out for another category, states, that of the 90 cases remaining, 62 were in males, 23 in females; in the remainder, sex not being mentioned. That the majority of cases have occurred between the ages of 20 and 40 years, "but that persons have been attacked as early as three and as late as 67 years of age." He makes three stages, (*a*) invasion, (*b*) paralysis, (*c*) convalescence, and says there is, as a rule, no fever. Fifty-three cases in Ross's table are due to causes such as operate in ætiology of peripheral neuritis, viz.: diptheria, septicalmia, alcoholism, syphilis, malaria, infectious fevers, metallic poisons, etc. In 37 the causes were uncertain.

In 47 cases out of the 90, no mention is made of paresis of muscles supplied by bullar nerves.

In 15 out of this 47, it is merely mentioned generally that the paralysis pursued an ascending course, or that the extremities were feeble. In 20, it is stated the lower extremities were first or most profoundly affected than the upper extremities. In ten cases, the lower and upper extremities appear to have been attacked simultaneously. In one case, the lower extremities were alone affected, and in another, the upper extremities were attacked before the lower; 52 died, all in stage of paralysis, 38 made partial or complete recovery.

Morbid anatomy.—38 of the 52 cases were examined post-mortem, as were three cases dying of intercurrent attacks of acute disease in stage of convalescence, and one that died of abdominal cancer; making in all 42 autopsies. The case of Bauchet and the two cases of Schultze are the three out of the 93 tabulated cases Ross does not include as ascending paralysis *per. se.* in deference to Schultze where cases lasted six months and seven months respectively before death took place, and who prefers the name in such cases of "ascending atrophic paralysis." As no autopsy was made in the cases the true pathology was not found.

Ross says, "To sum up, the medulla and spinal cord were subjected to microscopical examination in 26 out of 42 cases in which an autopsy was obtained; in 14 of these cases both were found completely normal; in four

[3] On Peripheral Neuritis, A Treatise, p. 91.

more the lesions were situated in the medulla oblongata and clevical region—a localization which affords no explanation of the symptoms — and in the eight cases remaining, the changes described were so slight in degree that they can not be taken as evidence of myelitis. Some of the nerve-roots or peripheral nerves were examined in nine only of the 42 cases, and in three of them were found normal; while in the remaining six cases degenerated nerve fibres and other evidences of neuritis were observed. The balance, therefore, of the evidence afforded by morbid anatomy is largely in favor of the view that the essential lesion is to be found in disease of the peripheral nerves and nerve roots, rather than in the nerve centres."

In this case then—No metals, organic or other poison being found leaves us in ignorance of the subtle agent that must be the actiological factor.

The alterations found in the blood too, were the result of the same cause. It must have been aptomanic or toxine, and when the rapid advances in bacteriology reach the stage of isolating these toxic substances, we may hope too, for cure by the administration of antitoxines.

There being but five cases of Landry's Paralysis recorded in patients over 60 years, the explanation may be from the fact that tissue resistance is greater in the system that has become more or less immune to infectious processes through its having combatted many; whereas in the young the severe symptoms are less resisted for the contrary reason and the patient succumbs.

May not the pathological process in the quickly fatal cases be the incipiency of organic change which the accurate microscopic "techinque" is yet too crude to detect?

And may not this case of neuritis, running much the same course as Landry's Paralysis at first, be one tided over; the beginning organic change then having an opportunity to already continue.

Since the above report was made an abstract of a paper (by Kirilzew and Mamurowsky read at the "Jubilee" Nervenheil und Psyschologie in November, 1890.) was seen in the *Journal of Mental and Nervous Diseases*, 1891, xviii. 154. The paper was on "A Case of Landry's Paralysis with Autopsy." Patient was a locksmith, twelve days ill, died with well marked symptoms of Landry's Paralysis. Acute parenchymatous neuritis was found in nervus of feet, legs, forearms; to a slight extent the roots of clevical and lumbar enlargements of spinal cord. Cord - hyperæmia of gray cloudiness and atrophy anterior ganglion cells and accummulation of cells in and about central canal. Most marked evidences of inflammation were in the peripheral nervus.

A CASE OF SYRINGOMYELIA.

By F. X. DERCUM, M.D.,

Clinical Professor of Diseases of the Nervous System, Jefferson Medical College.

IN view of the interest attaching to syringomyelia, and especially in reference to its pathology, the following case may not be void of interest.

D. A., male, white, aged 62 years, had formerly been a clerk; married, native of Connecticut. Was admitted to the Infirmary for Nervous Diseases, April 16, 1892. Service of Dr. S. Weir Mitchell.

The family history revealed that the father had died of phthisis, but his mother and other relatives had enjoyed very good health.

The personal history was practically negative up to thirty-seven years of age, when he states he was attacked very suddenly by extreme pain in the back, and his trunk and limbs became rigidly extended, his head being drawn back (opisthotonos). This condition, he says, continued for five weeks, and also that during the greater part of this time he was unconscious. It was impossible to obtain from him any more definite account of this illness. However, his convalescence from it was very tedious and for a long time afterward there was pain in the back, weakness in the legs and numbness of the hands. Later, recovery was apparently complete and he remained in good health for some five years, when he again noticed a numbness in the fourth and fifth fingers of both hands. This numbness began in the fingers of the right hand, but soon appeared in the left hand also. It increased somewhat in severity, but did not spread to the other fingers or other portions of the hand. It persisted, according to his statement, for some sixteen or seventeen years without further change. At this time, that is three or four years ago, he commenced to notice a sensation of "drawing or tightness of the tendons" in the arms. This steadily increased, and later on actual contraction of the arms made its appearance. Some

three years before this period he noticed a sensation as if the soles of the feet "were too much rounded." He says that he lost some of his certainty in walking. This steadily progressed until a marked staggering gait was attained. This continued until three years ago. The numbness also appeared in the feet and increased in degree, the condition being similar to that which already existed in the hands. His case had been slowly progressive, he thinks, and from bad to worse.

On his admission, the following notes of his condition were made by the resident, Dr. Pearse.

Present condition, April 16, 1892. Lies in bed with arms and legs much contracted. Examined for sensation, it is found that it is deficient in all the extremities. Voluntary motion is much impaired owing to the presence of the contractures. There is no ankle clonus, though the knee-jerks are exaggerated. Plantar reflex present. Pupils react well both to accommodation and light. Has lost control of both sphincters, bowels obstinately constipated. No apparent disease of spine, no pain elicited on pressure, or percussion. There is some pain in the hands. Character of this pain not made out very clearly.

Examined again, May 5, 26, 1892. Is able to move both legs and flex and extend them, but to a limited degree only. Has grown progressively weaker. K. J. appears to be normal on the right side, but is now absent on the left. Jaw-jerk not elicited. All of the muscle-jerks appear to be ++. There is a left-sided partial anæsthesia quite well defined in the middle line of the body. It is, however, most profound on the inner side of the left leg. It is now noted that he can distinguish heat from cold readily upon the right side of the body and upon the right leg, but on the right arm the power is evidently diminished. On the left side of the body and on the left arm and leg the thermal sense seems to be altogether lost.

June 15, 1892. Now, under the care of Dr. Dercum. The above notes as to the condition present corroborated. In addition, some wasting of both arms is noted, together with wasting in both legs, especially about the calves. Further, a bed-sore has made its appearance on each hip and in addition a very large one, five inches in diameter, over the sacrum. The knee-jerk is now absent on both sides. Tongue excessively coated. Patient is evidently in very bad condition; moans and groans almost con-

stantly, and especially at night. Upon being questioned he says that he suffers no pain, only a "general uneasiness." An examination of the urine reveals much pus and albumen, but no casts. A few days later the patient became delirious and sleepless, sedatives having little effect, and he finally died on June 28, 1892.

The autopsy was performed by Dr. C. W. Burr, Pathologist to the Hospital. It revealed among other changes of minor importance, a large cavity occupying the gray matter of the spinal cord. This cavity was most marked in the cervical region and to a less extent in the dorsal. It was absent in the lumbar region.

The microscopical appearances are as follows:

Microscopical examination of sections from the cervical region reveals extensive destruction of the grey matter and in addition sclerosis of both lateral tracts. The anterior cornua of the grey matter are to some extent preserved, though they seem to contain fewer cells than normally. Throughout the anterior cornua appear as though lopped off. The lateral processes if they exist at all are excessively distorted. Laterally and posteriorly the cavity encroaches much upon the general substance of the cord. Indeed, posteriorly the inroads have been so great that the posterior roots and root zones can with difficulty be defined. In some sections the posterior cornua seem largely to have disappeared. In the dorsal region, however, in which neighborhood the cavity is decided smaller, these parts are comparatively well preserved. In the dorsal region, also, the cavity is far more eccentric, being most marked toward the left side. In the lower dorsal and lumbar region the cavity is not detected. The walls of the cavity are exceedingly interesting, being made up of a richly nucleated, delicate connective tissue. That portion immediately lining the cavity is especially loose and delicate. Very soon, however, it becomes slightly more dense, but as we approach its periphery it again becomes more delicate and its fibres again separate more widely. In its peripheral portions are seen here and there numbers of nerve tubules. These, however, are absolutely wanting in the denser and more central portions. We are at once impressed with the difference which this connective tissue formation presents from that of ordinary sclerosis when we direct our attention to the sclerosed lateral tracts in the same sections. The latter tracts are replaced by connective tissue dense in character, presenting few or no

nuclei and differing strikingly in its physical appearance from the tissue just described. Further, the connective tissue formation lining the canal appears to pass from very fine gradations into the general substance of the cord. One is also impressed in examining these sections with the unusual vascularity of the cord in the immediate neighborhood of the cavity. The blood vessels are often numerous and large and sometimes quite tortuous. They do not, however, for the most part show any change in structure, though in a few instances they seem slightly thickened. Sections of the lumbar cord are of special interest as suggesting the possible sequence of the changes. As already stated, no cavity existed in the lumbar cord. Indeed, the central canal is entirely obliterated and in its place is found a rich deposit of nuclei, some within the lumen of the canal, but the bulk are aggregated about its periphery. Their arrangement, number and appearance closely suggest those found in the walls of the cavity of the dorsal and cervical regions. It is very probable, indeed, that we have here represented the beginning of a change which finds its completion in other portions of the cord.

A CASE PRESENTING HALLUCINATIONS OF SIGHT, TOUCH, TASTE AND SMELL.

By CHARLES S. POTTS, M.D.,

Instructor in Nervous Diseases, University of Pennsylvania; Assistant Neurologist to University Hospital.

W. B. H., aged 64 years, married and a clerk by occupation, presented himself at the Clinic for Nervous Diseases of the University Hospital, on November 12, 1892, and gave the following history, viz.: Family history good as regards nervous troubles excepting that one granddaughter is subject to chorea. The patient has never been a nervous man and has always enjoyed good health with the exception of a severe attack of grippe in 1890. He does not use alcohol, but chews considerable tobacco. No history of venereal disease. The present illness began one year ago (in 1891), and was preceded by an epidemic of fleas in the court room where the patient had his office. Several days after the fleas had been exterminated, while writing at his desk, he felt as he thought something biting him upon the legs, this he attributed to the fleas, and feeling the same sensations while at home, he had the entire house cleaned, carpets removed, etc., in a vain effort to get rid of the supposed insects. After this had continued for three weeks, one day while writing at his desk he saw what appeared to be a large black spider drop down in front of him. He put it, as he thought, on a shovel and threw it into the fire, but it constantly reappeared, and he moved his office from one part of the building to another in a vain endeavor to get rid of it. During this time the biting, which he likens unto needles being stuck into him, continued. After this, in rapid succession all sorts of objects appeared to him; for instance, at nights he would see four snakes, one at each corner of the bed, those at the head would be black, at the foot cream colored, with heads like bull frogs. During the day he would see little elephants, standing on their heads, rats with bread in their mouths, etc.

In January, 1891, he was compelled to cease work. The following March he ceased seeing the elephants,

etc., but they were succeeded by innumerable worm-like creatures, "as fine as a silk thread and of indefinite length, of different colors,—red, white, black, etc., some having human heads, others heads resembling that of a horse." These objects are continually crawling over him, getting into his food and crawling into his nose and ears; they have a "slimy taste and musty smell." At times he vomits his food on account of them. They continually bite him and he sees them get on other people. He is continually picking these objects off of him. In addition to this, the ground appears to be covered with smaller creatures, resembling small pieces of hay. The patient knows that these objects have no real existence and can reason why they could not possibly exist except to his own senses. Examination of the eyes showed slight opacities of the lens with retino-choroiditis. Urine was normal. Knee jerk is diminished, but it can be reinforced. The patient was only seen at the hospital three times, his last visit being December 23, 1892, from which time until a few days ago he has not been under my observation. The fact of these hallucinations being first apparently suggested by the visitation of fleas (an actual occurrence) which occurred just previous is, I think, of interest.

Examination[1] for sensory changes resulted as follows: Sense of touch well preserved. Painful sensations as the prick of a pin were felt as something hot, and when compared with a genuine hot object, were said to be the same sensation. Analgesia was present in a few scattered areas, especially on the arms. In a few places he claimed to feel two points as three. Temperature sense good. Testing his visual field for form and color, resulted as seen in Fig. I.; it is seen that the form field is contracted and with the exception of in a few points is inside the field for red. The blue field was not taken. The fields were taken by Dr. Jas. Wallace, chief of the Eye Clinic of the University Hospital, and were verified by several trials. While at the time of the patient's first visit, hysteria was hardly considered, as there was nothing in the man's behavior and appearance pointing that way, the subsequent history since I saw him one year ago, and which is detailed in the discussion, together with the sensory changes above noted, would seem to point towards this probably being a remarkable case of that disorder.

[1] This examination was made after exhibiting the case to the Society.

Stated Meeting, December 26, 1893.

President, Dr. CHARLES K. MILLS, in the chair.

Dr. FRANCIS X. DERCUM exhibited

A CASE OF SCLERODERMA OF TRAUMATIC ORIGIN.

The patient whom I have here is one of great interest as the affection is exceedingly rare. Last August this man, while working in a stooping position, was struck on the head by a piece of ice weighing sixteen pounds which had fallen a distance of ten feet; he was rendered unconscious and brought to the Jefferson Hospital. There was a transverse lacerated wound over the occiput which was sewed. The wound did well surgically, but the man himself did not do well. He gradually became more and more neuræsthenic and had persistent pain in the neighborhood of the scar. He has also general headache and backache and some tenderness over the spine.

In addition to the symptoms of general neuræsthenia, we noticed four weeks ago (what the patient had observed two weeks earlier), a curious condition of the skin. The skin about the arms, shoulders and neck began to feel very tense and puffy. Dr. Cantrell has studied the case with me and the condition of the skin has steadily became more marked as we have watched the case. The case is evidently one of scleroderma. This affection in a number of instances has followed nervous shock. In the minds of dermatologists, scleroderma is at present closely associated with morphœa and hemifacial atrophy. The later condition we are pretty certain from one or two post-mortems is due to descending degeneration of the motor root of the fifth nerve, In this class of cases we evidently have to do with a tropho-neurosis.

Dr. J. ABBOTT CANTRELL showed

A CASE OF MORPHŒA OF TRAUMATIC ORIGIN.

Dermatologists look at these cases as two varieties of one disease. One is the diffuse or œdematous form and the other the circumscribed or atrophic form. I have brought this case because Dr. Dercum intended to report his case of diffuse scleroderma and because, as in his case, there is also a history of traumatism. This child while playing at school fell and hit her head on the sharp edge of a desk. There was no rupture of the parts. There was swelling and the consequent œdema following such accidents, and the atrophied condition which you see began immediately afterwards. The injury dates back about eighteen months. This is the form which in the books is called morphœa. In this case it affects the right side of the head extending down towards the external canthus of the eye.

Dr. CHARLES K. MILLS.—In the first case it seemed to me that the pathological condition probably involved the subcutaneous tissue as well as the skin; in other words, we may have not only scleroderma, but a myxœdemoid condition.

The second case can be readily explained on the theory of traumatism involving the motor and trophic branch of the fifth nerve in the particular locality where the affection began. I have seen several cases of hemiatrophy of the face. I believe that in all these cases there is involvement of the muscles supplied by the fifth nerve. I might call attention to a paper by Montgomery that appeared in the *Medical News* during the past year on unilateral facial hypertrophy.

Dr. JAMES HENDRIE LLOYD.—I showed before this Society, about a year ago, photographs of a case of scleroderma or hemifacial atrophy, beginning well up on the vault of the cranium, running down the median line of the forehead and involving the root of the nose, running down also under one eye. In my case, however, there was no history of trauma. The patient was a young Swedish woman at family service. There was no atrophy of the muscles, nothing but this localized area of atrophy.

Dr. THEODORE DILLER, Pittsburg.—My experience with myxœdema is limited to two cases, the second of

which I saw only a short time ago. In examining Dr. Dercum's case, the question arose in my mind: in what respect does this skin-thickening differ from that which is seen in myxœdema? Another query that arose was: might not this possibly be the beginning of myxœdema? I simply raise these questions with the hope that Dr. Dercum will consider them in any further remarks that he may have to make.

Dr. FRANCIS X. DERCUM.—I dismissed the idea of myxœdema on account of the absence of other symptoms—the absence of the spade-like hands which are among the earliest signs of true myxœdema. The face was not affected early. I had the opportunity of seeing the man before the condition came on and have seen him frequently since. Lastly, the case has followed the history of scleroderma which usually begins over the back of the neck and extends to the trunk and limbs.

Dr. Cantrell's case is of exceeding interest because it looks as though the blow might have given rise to some direct nerve injury and the atrophy might be looked upon as the result of that. If this is the proper explanation it is curious that these conditions do not more frequently follow nerve injuries.

Dr. C. S. POTTS exhibited a patient with

HALLUCINATIONS OF SIGHT, TOUCH, TASTE AND SMELL. (See page 40.)

The patient stated that in January, after he had seen Dr. Potts, he passed into a condition of profound stupor from which he was aroused only by severe shaking and loud calling. This condition continued from the last of January until the fifth day of March. The latter part of February he was removed to the asylum. At the end of March he was discharged from the asylum.

The patient then retired.

Dr. H. R. SPRUANCE, of Wilmington.—In regard to the attack of unconsciousness to which the patient has referred the family give a different history. While he says that he was asleep, his family state that at one minute he would be so weak that he could not move, the next he would pick up his wife and carry her, and at times it required two or three men to hold him down. He was taken to the Delaware State Hospital for the Insane. There he was treated with tincture cinchonæ

comp. and syrupi pruni virgin., and got somewhat better, ordered by Dr. Richardson now Superintendent at Norristown, under whose care he was, before he was sent to the hospital. He had a nurse with him at night constantly for one week, but says he has not yet seen that nurse.

He describes one of these objects as red, pushing a red hot iron around the room and across the bed and with an explosion coming down and lighting on him. Others are red and white.

Dr. JAMES HENDRIE LLOYD.—The French authors say that the reason that the English and Americans do not have hysteria is because they do not diagnose it. The longer I live and the more I see of the practice of medicine, the more am I convinced of the accuracy of this remark. There is but one disease that will produce the lethargy such as has been described in this case. There is no disease except the grand hysteria that will do this. That lethargy is one of the most marked psychical stigmata of the grand hysteria. I do not know of any other condition that will produce a lethargic state for four or five weeks in a healthy man unless it is hysteria in one of its major manifestations.

In *Brain*, Hitzig, of Halle, one of the most eminent experimenters in Germany, has put on record a case of remarkable lethargy occurring in a workman twenty years of age, following extensive injury to the head and arms. This lethargy presented a periodical aspect. The man would lie for thirty-eight or forty hours in the lethargic state, all the time losing weight and the specific gravity of the urine running down, the nitrogen being decreased. In other words, showing the peculiar alteration in nutrition which has been described by Tourette and Cathelineau, this being preceded by hysterical prodromes. The paroxysm would pass off, to recur in nine or ten days. This had continued for several months when the man came under Hitzig's care and was hypnotised and cured.

This lethargy is always associated with intervallary stigmata. I suspect that this man, if carefully examined, would have shown some of these stigmata. There might have been found some alteration in sensation, probably some areas of anæsthesia, or some narrowing of the visual fields. He has a vision of red and probably has preservation of the red field which the French for the last twenty years have been talking about as one of the most common visual stigmata of hysteria.

Charcot has described one peculiar characteristic of the grand hysteria which he named zoöpsia, the hallucination of the sight of animals. This is seen in a minor degree in many cases of hysteria. I have, however, never seen or heard of a case where this was so marked as in this instance. Considering the zoöpsia, considering the lethargy and considering the slight tremor of his hand which is distinct, I have very little doubt in my own mind that this is one of the major states of grand hysteria, one of the aberrant, atypical forms which we see not infrequently if we look for them.

There are, it is true, certain sensory hallucinations in the insane. I do not think that this case can be included among these. A few years ago, I saw at the Philadelphia Hospital a colored man in an advanced stage of senile paresis. He had the hallucination that he was covered with a serpent's skin with two holes for the eyes, one for the mouth and another to urinate through. This general cutaneous hallucination we see in senile paresis. The present case is advanced in years, but presents no evidence of insanity. I do not think that we can hesitate as to the diagnosis.

Dr. THEODORE DILLER, of Pittsburg.—I might, in this connection, relate two cases of lethargy which have recently come under my observation. A colored boy, 14 years of age, went to Washington three years ago, and while there got into a fight and was struck on the back part of the head with a heavy iron bar. A wound three or four inches in length was produced. He was rendered unconscious for a few mintues. The wound healed and he was apparently well for six months. It was not thought a fracture had been produced. Then he had an attack of lethargy which was frequently repeated. He got stupid and heavy and would lie in bed for eight days, passing the urine and fæces in bed and taking scarcely anything to eat. In fact, the case was almost like that one reported by Professor Hitzig. These attacks recurred every four weeks and always lasted precisely eight days. After this had gone on for two years he returned to Pittsburg and came under my observation. I found concentric contraction of the visual fields, and some anæsthesia and great susceptibility to hypnotism and concluded that he was an undoubted case of hysteria. A full account of the case will appear in the next number of *Brain*.

The second case was a very extraordinary one of hys-

terical motor aphasia and lethargy. The man came under my observation two months ago. Mentally, he seemed stupid and child-like, but that, I imagine, was his usual condition. He stated that his voice would suddenly leave him and then come back. These attacks would occur every two or three days and would continue from three to six hours. During this time he would be perfectly conscious and be able to understand conversation, recognize objects and think of the words that he wished to use and write them.

Clinically, we would say, that this was a case of motor aphasia; but from the fact that the attacks came on suddenly and disappeared and reappeared suddenly, there would be *a priori* reason for thinking that the case was one of hysteria. On examination, I found general blunting of sensation all over the body, but now special spots of anæsthesia. There was concentric contraction of both visual fields. A month later I found him in the insane asylum. There he had no attacks of motor aphasia, but was having attacks on alternate days, lasting two or five hours in which he would be apparently unconscious, and from which he could not be roused. I have no doubt that these are attacks of lethargy. This man possesses great faith and has experienced great comfort from blessings received at convents. A strong suggestion followed up on several occasions caused these attacks of lethargy to disappear. The man is now brighter and improved in every way.

I must agree with Dr. Lloyd that lethargy is a hysterical manifestation. I know of no other affection of which it is a symptom, and it can scarcely be regarded as a disease in itself.

Dr. JOHN K. MITCHELL.—In spite of the arguments that have been advanced, I am not convinced that this case is hysterical. If, on examination, it were shown that he had narrowing of the visual fields and reversal of the color fields, I, perhaps, should think that this was a hysterical condition. Still, it seems curious that it has not been mentioned that so many of the cases of delusions of uncleanness take the form that they are covered with animals. I have had for a year an insane woman under my care who has the delusion sometimes that she is covered with black insects and sometimes with caterpillars. She will pick them off and throw them on others. We have gotten rid of the insects for two weeks at a time by the subjective measure of scrubbing with oil. This

woman has only two delusions, the one of uncleanness and another that her husband has lost his money and they must pull the house down. In some respects she is perfectly sane. In my opinion this patient presented is in the early stages of insanity and will grow worse as time goes on.

Dr. JOSEPH LEIDY, JR.—The fact that the man recognizes that these objects do not exist and presents no other evidences of insanity is against such an idea. In connection with Dr. Mitchell's remarks, I remember distinctly a patient as a neighbor at table at the Pennsylvania Hospital, a man who was perfectly sane upon every subject except one, that of uncleanliness. He had periodic attacks of melancholia. At other times, the mesaphœbia took the place of the insanity.

In this case, I think that until the visual fields have been examined and the question of anæsthesia determined in connection with any other symptoms, we should reserve a positive opinion, although it favors hysteria.

Dr. CHARLES K. MILLS.—This man may pass into a condition of true insanity. A patient may for a long time struggle against such hallucinations and finally come to believe in their reality. I remember the case of a man who for months had notions partly hallucinatory with which he struggled for a long time, feeling that they were not real, but at a certain time, which he described, he became convinced that he had been in error and that these were realities. From that time he continued to believe in them and out of a comparatively simple condition evolved a rather elaborate systematised delusion. Under the ordinary rules of diagnosis, Dr. Lloyd's position is well taken and this case could be better diagnosticated as hysteria than as anything else, but this fails fully to explain the case and fails to indicate with any certainty what the future of this man will be. We should do something in the way of treatment for this man. The strongest efforts should be made as the result of this consultation, by the physician in charge, and that it should be impressed on the patient, that his is a nervous affection that can be cured and that as a result of this consultation, means of cure have been decided upon. Then I think that it matters little what will be the exact method of treatment. Isolation, hydrotherapy, static electricity and metallic tonics would be of service.

Dr. G. BETTON MASSEY.—It seems to me that the

diagnosis of hysteria is hardly tenable. In the similar instance referred to by Dr. Mills, it can be seen that the case was one of genuine hallucinations, originally, in which the intellection of the patient failed to be overcome. I think that I have seen such cases where ultimately, as the hallucinations increased, the disease producing the hallucinations increased, the intellect became affected and the patients finally believed that the hallucinations were real. This man tells us that he does not believe that these things are real, but he acts upon the belief that they are real more thoroughly than would be the case in hysteria. He is constantly going through motions which to my mind are more elaborate than mere hysterical conditions would produce, for it is rare that hysteria acts itself out thoroughly. It seems to me that the best interests of the man and his family would be subserved by considering this a case of threatened insanity, and treating it from that point of view.

Dr. F. X. DERCUM read a paper on

A CASE OF SYRINGOMYELIA WITH AUTOPSY.

DISCUSSION.

Dr. CHARLES W. BURR.—I made a few weeks ago a post-mortem in a case of syringomyelia in which there were no sensory changes. The man began sixteen years ago to have pain in the neck, very severe and soon after began to lose power in the arms and slightly in the legs with some wasting. Six years ago he came to the Home for Incurables with paralysis of arms and legs. Sensation to heat and cold was normal and there were no sensory changes when I last examined the man, a year ago. The absence of sensory changes made were put to one side the diagnosis of syringomyelia.

At the post-mortem examination, I found quite a large canal beginning in the uppermost part of the cervical region and extending down to the fifth dorsal nerve and transversely occupying almost the whole of the cord, but microscopically not appearing to involve the posterior white matter very much. There has not yet been time to make a microscopical examination. The idea that sensory changes are an essential part of syringomyelia seems from this case to be misleading.

Dr. FRANCIS X. DERCUM.—The symptoms that would

ensue would depend largely upon the portion of the cord destroyed. There would be muscular atrophy if the anterior portion was encroached upon. In this case there was no marked anterior wasting. I am sorry that in this case I did not make more detailed investigation of certain muscles and certain muscle groups. There was, however, no marked wasting anywhere. The cavity was accentuated posteriorly as it was also on one side.

The case was of interest on account of the marked contractures. I have now under observation a case where there is marked atrophy, distinct thermal changes and Charcot joints. In the case reported to-night there were also contractures of both arms and legs. The cavity was very large in the cervical region, cutting off both pyramidal tracts. The patient had in addition to the symptoms of syringomyelia, those of marked lateral sclerosis.

Dr. JAMES HENDRIE LLOYD, read a paper on

PROGRESSIVE MUSCULAR ATROPHY WITH ARTHROPATHIES OF THE KNEE JOINT, (WITH EXHIBITION OF PHOTOGRAPHS OF THE HANDS AND KNEES.)

This case is of peculiar interest because it is one of pure muscular atrophy with arthropathies. The case came under my care at the Methodist Hospital where I found the woman suffering with enlargement of the knee joints. My predecessor and one of my surgical colleagues had tapped the joints. There was not the same lateral deviation as occurs in some cases. In this case, the attention had been directed almost exclusively to the knees. The moment that I saw her I was struck with the peculiar appearance of her hands. She had the main-en-griffe on each side. I found the arm muscles extensively atrophied, those on one side almost entirely gone. There was also scoliosis. I at first thought that the case was one of syringo-myelia and made suitable tests for sensation, but the results were negative. There was no thermal anæsthesia and no alteration of sensation in any way. I thought that I was not justified in making a diagnosis of syringomyelia. We have here the muscular, motor, atrophic, joint, and skeletal changes associated with syringomyelia, but not the sensory changes. I,

therefore, report it as a case of progressive muscular atrophy associated with arthropathies. At the same time, I think that the association of spinal arthropathies gives the case a rather unique appearance and that the condition is probably due to some form of gliomatous change in the medulla spinalis.

In a former somewhat extensive study of the subject I found arthropathies reported as occurring in locomotor ataxia, general paresis, syringomyelia, and sclerosis in plaques. There are no cases recorded with progressive muscular atrophy, except one case reported by Rosenthal twenty years ago. That case, I think, was likely one of syringomyelia. The present case will be reported more fully in detail for publication.

Dr. FRANCIS X. DERCUM.—We are still in the developmental stage of our knowledge in regard to syringomyelia and it may be that in these joint changes we have a point of differential diagnosis between simple muscular atrophy due to chronic polio-myelitis and muscular atrophy due to syringomyelia. Of course, it is only by the study of many cases that we can gain any definite knowledge upon this subject.

Dr. F. SAVARY PEARCE reported

A CASE OF MULTIPLE NEURITIS SIMULATING LANDRY'S PARALYSIS, IN THE RAPIDITY, ORDER AND EXTENT OF PARALYSIS. (See page 25.)

DISCUSSION.

Dr. CHARLES K. MILLS.—The question suggests itself whether the term neuritis as descriptive of this case is well chosen. Pain and sensory changes were not present, with the exception of slight tenderness in the popliteal space. The question occurs in connection with this case and other somewhat similar cases. Have we inflammatory disease of any type? Have we not really a form of toxic disease causing multiple degeneration, and should we not add to our nomenclature multiple degeneration of nerves instead of always speaking of multiple neuritis?

Dr. FRANCIS X. DERCUM.—I had an opportunity of studying this case during the summer and I was never absolutely certain in my own mind with regard to the

diagnosis. I roughly grouped the case under the head of Landry's palsy, although in some respects it differed from the latter disease. It looked as though the trouble, possibly toxic, had affected not only the cord, but also the peripheral nerves—as though it were a mixed case. I remember that it was Dr. Mills who first called attention to these mixed cases of multiple neuritis with cord involvement.

POLIO-ENCEPHALITIS SUPERIOR ACUTA; WITH REPORT OF A CASE.

By SAMUEL WOLFE, A.M., M.D.,

Clinical Professor of Nervous Diseases in the Medico-Chirurgical College, Philadelphia.

B. C. D.; male; æt. 39; married; driver of milk wagon. While following his usual occupation during September, October and November, he had occasional attacks of vertigo, occurring about once a week, so severe that he was obliged to grasp a support to keep from falling. During the same period of time he also had sensations mainly in the limbs, which he describes as of an itching character, and also some feeling of numbness. In the early part of December, the attacks of vertigo became much increased in frequency, so that he had several daily, and his wife says that at this time she also noticed that he squinted, the left eye she thinks being turned inward. On December 6th, he called on his family physician, Dr. Snively, complaining some of diplopia, giddiness, frontal headache, and staggering gait. He still continued to work, and on December 13th called again on Dr. Snively, when it was noticed that the eyes were immobile and fixed in the mid position, the facial muscles had lost power and tone, and the speech was much altered. He, however, still persisted in going over his milk route daily, until, on the 15th, when in one of his vertiginous attacks, he fell out of the wagon. He then went to bed, and Dr. Snively was called next day, finding a temperature reaching 100.5; pulse increased in frequency to about 100; considerable somnolence, and a little mental disturbance, which was thought to amount to a few hallucinations. There was no activity of the bowels, and a small cathartic dose of aloin ($\frac{1}{5}$ gr.) was administered, which purged severely and continuously for an inordinate length of time. After a few days of slight fever there was free sweating, and the pulse and temperature became normal. There was about this time, some sweating of the left side of the face while the right was dry. A considerable degree of cutaneous anæsthesia of the extremities, the legs and

forearms especially, was made out. I was called to see the patient on the 23d of December, seventeen days after he had been first seen by Dr. Snively.

I found a partial ptosis, which would probably have been complete but for the paralysis of the antagonists to the levatores, the orbicularis palpebrarim; entire immobility of the eye-ball in every direction, the eye being fixed in the mid-position, and slightly bulging; loss of power to accommodate; great dilatation of the pupil, entirely irresponsive to light (a lighted candle could be brought almost in contact with the eye, without producing the least contraction). All these symptoms were perfectly bilateral. There was also complete bilateral facial paralysis, so that he could not raise his eyebrows, close his eyes, could not blow or whistle. He found eating and drinking difficult on account of the tendency for the substances to escape through the toneless lips. The speech was nasal, muffled, and guttural, partly from paralysis of the velum palati (the uvula deviating very slightly to the left) and largely from paralysis of the levatores alæ nasi, which was shown when he pushed up the nasal cartilages during phonation, his speech then being markedly more distinct.

Smell had been lost according to his statement, during the first few days he was in bed, but had already been recovered. Taste also had been completely lost, but now also had partially recovered. I found on testing that on the left side of the tongue, taste was normal on the anterior part, and on the posterior part, paræsthetic. He would mistake salt for sugar, or vinegar for salt, and so on. On the right half of the tongue the sense of taste was wholly absent.

He had a feeling of numbness in both arms and legs combined with a "pins and needles" sensation. I found decided but not complete anæsthesia and analgesia on the outside and back of right leg, and on the inside of left, as well as on the ulnar and posterior surfaces of both forearms. There was great pain on pressure over the fourth lumbar spine, and also some spontaneous pain in this region.

The plantar, the cremasteric and the abdominal reflexes were all diminished, especially the plantar on the left side. The knee jerks were absent, even on reinforcement. There were spontaneous movements on holding out the limbs and a coarse tremor of all four extremities on voluntary motion. He sways on standing

with eyes open, very much more with eyes closed. His gait is very irregular, and ataxic, especially marked on turning in his course. No marked loss of power could be determined in any muscle, or set of muscles, in the extremities.

He has never had syphilis, and never used alcohol; he chews tobacco excessively and smokes some. He has an aunt living who is paralytic, and another aunt and an uncle have died of paralytic affections.

My friend, Dr. J. A. Cramp, was asked to examine the eye grounds, and to test the visual fields. He reports as follows:

My dear Dr. Wolfe:

I examined Mr. Daniels as you requested. External examination showed complete paralysis of all the ocular muscles (bilateral), the pupil was dilated and would not respond to light. Accommodation was paralyzed. There was ptosis principally on left side; the patient's distance vision was fair, being $\frac{6}{9}$; no contraction of the field of vision. The ophthalmoscope showed no fresh zones of inflammation in neither the retina, choroid, nor nerve; although the nerve borders above and below were slightly obscured by connective tissue; this condition I would state, we often find in hypermetropia, which defect the patient had to the extent of about two dioptries.

On the 3d of January, eleven days after the above condition was found, I found that the pupil responded readily to light impressions and was not greatly dilated. Power of accommodation had returned so that he could read. The ptosis had largely disappeared. Taste had fully returned, and the anæsthesia of the legs had disappeared, but persisted in the arms. His gait was markedly less ataxic. The other symptoms were unchanged, except that a very slight improvement in his speech may be noted.

On the 19th of January, twenty-seven days after my first examination, I found the ptosis entirely gone, the pupils normal in size and reaction, a little spasmodic lateral movement of the eyes, usually of one alone, on attempting to follow an object. He could now raise his eyebrows, shut his eyes, blow, whistle, retain food within his lips, and could give some facial expression to his emotions, although in this respect, power was not proportionate to that of voluntary motion of the facial

muscles. The anæsthesia has disappeared, but there is an irregular paræsthesia of trunk, which he defines as a feeling of a hot cloth, lying on a patch of skin, now on the abdomen, now on the chest, and again on the back, but not constantly present in any situation.

He now stands well with eyes open or closed, but still is slightly ataxic in gait. There is slight inco-ordination of left arm, shown on touching his nose, and a coarse tremor of all four extremities. The left knee jerk is very slightly present, and develops into an almost normal one, on reinforcement. The right is still altogether absent.

On the 22d of January, when seen for the last time before the conclusion of this paper, he could move his eyes inward, upward, and downward to a considerable extent, but outward movement was still entirely absent. He could now smile broadly, and walk almost without any noticeable inco-ordination, with the exception of a halt before starting, in turning half way round. The sixth nerve seems the last to recover, and here there will probably remain some residual paralysis.

The treatment pursued in the case from the time I saw him, was potass iodid gr. × t.d., until about the third week, when $\frac{1}{50}$ gr. doses of strychnia sulph. were given in addition, three times a day. He was kept in bed, and all mental excitement and exercise were prohibited.

The chief, if not the exclusive lesion in this case, I believe was in the nuclei of the 3d, 4th, 6th, and 7th cranial nerves. The paræsthesia of the extremities, which existed for three months before the cranial nerve symptoms appeared, and the occasional attacks of vertigo that appeared during the same time, point to the possibility of some vague degenerative disease in its earlier stages, but the anæsthesia and analgesia with the ataxia, which appeared in conjunction with the major symptoms of the main attack seem to me to point to involvement of the tegmentum, or probably the thalamus. This involvement seems to me to be indirect, most likely due to circulatory or nutritional disturbance of some kind, while the direct lesion was inflammatory in nature, and in-

volved the motor cells of the nuclei of the nerves already mentioned, just as an anterior polio-myelitis has the inflammatory action limited to the motor cells of the anterior cornua. Spasm and ataxia manifested in the gait, it should be remembered, were symptoms which entered constantly into the clinical picture of polio-enciphalitis superior acuta, as observed by Wernicke, and their association in the present case with a history of paræsthesia, affecting the same parts for some time previous to their development, might be looked on as purely incidental. The topographical relations of the nuclei of the 3d, 4th, 6th, and 7th nerves to the tegmentum, are such that a focal lesion of the former, might be expected to affect the latter in such a way as to produce indirect symptoms pertaining to sensory conduction, while the crusta and consequently the pyramidal tract might easily escape. Again, a focus of inflammation involving the nuclei, would most naturally involve also the contiguous parts, and thus the symptoms might be direct. The order of recovery, however, the sensory phenomena being the first to disappear, argues in favor of the former view.

As to the nature of the lesion, the view that it was inflammatory is tenable on the ground of gradual onset, slight rise in temperature and acceleration of pulse, and the occurrence during the prevalence of an epidemic of influenza. I need not dwell upon what has been a matter of such universal observation, the tendency of the toxine of *la grippe* to produce lesions of both the peripheral and central parts of the nervous system. That some such agency is active in the production of acute anterior polio-myelitis, and probably of some other acute system diseases of the nervous tissues is a theory which is undoubtedly gaining in favor from year to year.

The transient loss of smell, and the somewhat more persistent loss of taste, are not very easily explained. It may be worth while to consider that the uncinate gyrus, the hypocampal gyrus and the anterior commissure and cornu ammonis are all parts whose topo-

graphical relations to the nuceli certainly affected, are such that they sustained more or less liability of being indirectly, if not directly, affected. The transient nature of these symptoms, too, was such that, it may be assumed that structures of a different nature, as the cortical cells, were affected, or that the symptoms were of an indirect nature. In regard to the affectation of taste, the view of Gowers, that all the taste fibres arise from the nucleus of the fifth, must be borne in mind. The absence of all other sensory and motor symptoms connected with this nerve, however, miltitates strongly against this view of the nature of the affection. Somewhat more plausible argument may be made in favor of an implication of the fibres of the chorda tympani, through nutritive changes, brought about by their close relation to the facial fibres, proper. That these fibres (the facial) underwent trophic changes of a decided character is, of course, simply in accordance with one of the facts most thoroughly known, in the pathology of nerve degeneration. That such intense changes might exercise a deleterious effect upon other very closely associated structures is by no means an altogether unwarrantable assumption. I, however, incline to believe that in the affections of both taste and smell the origin was cortical rather than nuclear or peripheral.

The diplegia facialis is a symptom, which, heretofore, has not been observed in this disease. When we consider the close relations of the nucleus of the 6th and 7th nerves, there being most probably communicating fibres between the two, and the still closer relation of the root fibres of the facial to the nucleus of the 6th, it seems rather remarkable that it should be the rule that the facial escapes. The probable origin of the fibres which supply the levator palpebræ, from the nucleus of the 7th establishes a still further close relation between the occulo-motor nerves and the facial. It will be seen, then, that the present case is, in all respects, one of the most completely typical yet observed. The involvement of the three branches of the facial, since we are able posi-

tively to exclude a peripheral lesion, at once places this focus in the nucleus. The complete and persistent loss of power of expression, also, fixes the lesion below the optic thalamus, unless, indeed, we may consider that organ implicated. At all events, the cortex and the fibres of the corona radiata, as well as the intra-capsular course of the tract can positively be excluded.

The completeness of the ophthalmoplegia at once removes the disease from the chronic type which has been so well studied by Von Graefe. In this the pupil constrictor and the ciliary muscle are not involved. Besides this we have a series of concomitant phenomena, including a specific poison, acting as a cause which place the disease in the acute class. The order of recovery is interesting, since those symptoms disappear first, that never appear at all in the chronic variety, indicating that a less intense cause is sufficient to affect the extrinsic muscular apparatus, than would suffice for the intrinsic.

The tendency to recovery in this case, is also peculiar, since the type described by Wernicke is almost invariably fatal. There is, however, in this, a still further analogy to the prototype, acute anterior poliomyelitis, which again counts in establishing this case as particularly typical. Should there remain, as I think there will, some residual paralysis, this observation will be still more strongly confirmed.

The study of this disease has been so much limited, that its existence as a pathological entity is doubted by many neurologists. What observation there has been is probably confined entirely to Germany. Instances, therefore, are rare. The present case, it would seem, should go far in establishing its rank as an independent disease. The chronic progressive variety occurs more often, and its existence and identity are well established.

To summarize and conclude: I have in this paper reported a case of inflammation of the motor cells of the nuclei of the 3d, 4th, 6th and 7th cranial nerves, of infectious origin, in which the initial symptoms were diplo-

pia, strabismus and vertigo; the established symptoms directly due to the lesion, external ophthalmoplegia, cycloplegia, iridoplegia (mydriasis) and ptosis with diplegia facialis giving rise to great alterations in speech; and with loss of smell and taste, inco-ordination, and sensory disturbances as indirect symptoms; the entire complement of symptoms being established in the course of less than three weeks, after a duration of about ten days progressive recovery; classified as polio-encephalitis superior acuta, presenting probably the most completely typical case yet recorded.

1624 Diamond Street, Philadelphia.

TWO CASES OF SYRINGOMYELIA; ONE OF UNILATERAL TYPE.

From the Polyclinic Service of CHARLES K. MILLS, M.D.,

Philadelphia.

Reported by J. W. McConnell, M.D.

CASE I.—R. A. R., white, male, aged 52 years, an iron roller, has a good family history. He denies venereal taint, does not use tobacco and indulges but little in alcoholic stimulants. His physical history is good up to the time of commencement of the present trouble, having had no illness or condition requiring surgical attention, excepting a very painful whitlow with which he suffered about twenty years ago. The patient has been an iron worker since he was twelve years old and is a hard working man.

About twenty years ago, he was troubled with pains, shooting in character, occurring in the right shoulder or arm or hand separately, and at times involving the different portions of the upper extremity simultaneously. He never had shooting pains elsewhere and for many years has not been conscious of pain of any kind. There is no history of diplopia, vesical or rectal trouble, nor of ataxia.

Nine years ago (1885), his attention was drawn to a peculiarity in his sensibility to heat and pain. While at his work he leaned against a hot steam pipe and burned the right side of his back severely without being conscious of it, and he was unaware of anything having happened until two weeks afterward, when the bloody bedclothing caused him to wonder as to the source of the blood. Again last winter (1892), he burned himself close to the seat of the first burn by resting against a hot furnace plate. Of this injury he was ignorant until his wife, seeing the ulceration, inquired the cause.

Both burns were very slow in healing, and at the present time a mass of proud flesh marks the seat of the injuries.

The present condition of the patient is as follows: He is a well nourished man, whose back just below the angle of the right scapula, is marked with two large scars,

each about four inches long and two inches wide, the results of the previously mentioned burns. He complains of no pain, no loss of power, no feeling of ill health, nothing but a loss of certain sensibilities, which loss makes his employment a dangerous one.

On the right side of his body, commencing just under the jaw of that side, is noticed a peculiarity in the sensibility to heat. This peculiarity is not a complete loss of thermo-sensibility, except in certain localities. In portions of the back, right arm and leg, is complete loss, in others a retardation of heat sense, in a third the sensibility to heat and cold seems to be confused.

Over these same areas is also a change in the sensibility to pain. Here, again, we have not a complete loss, but rather a retardation in the conduction of painful sensations. Sticking a needle into the right side of the body does not cause pain, but if the needle is allowed to remain in the flesh a few seconds the patient can differentiate between sharp and dull.

Tactile sense does not seem to be impaired. He responds almost immediately to the touch of the finger. Muscular sense is also apparently normal.

There is no atrophy or loss of power in the affected side. Electrical examination gives negative results. Knee jerk and muscle jerk are decidedly plus on both sides.

CASE II.—S. C. S., white, male, aged 65 years, a farmer, came to the Polyclinic Hospital Clinic for Nervous Diseases. His family and personal history are negative in relation to any cause for the present trouble, and until seven years ago he had been free from serious sickness of any character.

In 1886, the patient noticed for the first time some weakness of the right lower extremity, commencing, as he says, in the foot and gradually involving the whole limb to the hip. In consequence of this weakness, he was required to use a cane to assist him in locomotion, and with that aid he could move about with but little difficulty. There was no pain accompanying this weakness at first, but in 1890 the patient became conscious of some uneasiness and stiffness in the limb, and later on had positive though slight pain in the lumbar region.

In 1889, the right knee became swollen, and the joint has gradually changed in shape and position. At this

time it was noticed that the muscles above the knee joint were wasting somewhat.

There is no history of urinary or intestinal trouble until 1891, when he commenced to have difficulty in passing his urine. No pain, but the bladder seemed to have lost in muscular tone, and the expulsive force was less than normal.

His condition January 25, 1893, was as follows: Intelligent, well-nourished man, with well-formed trunk and arms, but a peculiar deformity of the right leg. About the knee of this side is much wasting of muscular tissue, and the joint is so changed in shape that the limb presents the appearance of an arc of a circle with the convexity backward. When the patient walks he is able to bend the knee, but the weight of the body resting on that limb causes the arching or curving of the limb to be even more marked than when standing quietly on both legs.

The left limb is normal in appearance and presents no changes of any kind. Movements of the right foot are all impaired, but not totally lost. Those of the left seem to be preserved.

Flexion and extension of the right toes seem to be better preserved than any other movements. The muscles of the thigh, Iliacus, psoas, quadriceps, abductor, sartorius and glutei are partially paralyzed, as are also the gastrocnemius and soleus, while the anterior and posterior tibials and the peroneal group are almost completely so.

The movements of the right upper extremity are not impaired. Fibrillary tremors, however, are found here and also in the leg.

Reflexes in the right lower extremity are increased, but no change is found in those of the trunk upper extremity or opposite side. Foot clonus on the right side is marked and persistent. To electrical currents the muscles of the right side respond, but not so vigorously as those of the left.

Thermo-anæsthesia is noted from the fourth rib, on the right side, down the trunk and left leg, irregularly across the right shoulder and down the arm of that side. In certain localities, above the right nipple and just above the right groin, the loss of temperature sense is less marked.

The condition of the back is similar to the front.

The ability to differentiate between the sharp and

dull points of the compass is almost entirely lost over the areas of thermo-anæsthesia, and the loss seems to be more marked in those points where the thermo-anæsthesia is most decided.

Tactile sensibility is somewhat impaired, but by no means lost.

Stated Meeting, January 22, 1894.

The President, Dr. CHARLES K. MILLS, in the chair.

The PRESIDENT made some

REMARKS ON THE HISTORY OF THE PHILADELPHIA NEUROLOGICAL SOCIETY DURING TEN YEARS.

Dr. J. W. MCCONNELL reported from the Polyclinic Service of Dr. Mills,

TWO CASES OF SYRINGOMYELIA, ONE OF UNILATERAL TYPE. (See page 61.)

EXHIBITION OF THE UNILATERAL CASE.

DISCUSSION.

Dr. CHARLES A. OLIVER.—I have examined this man and find that the pupils are of the same size and that each iris responds equally and freely both separately and combinedly to light stimulus. I also find that they respond freely and equally in separated action to similar acts of accommodation and convergence, and combinedly to conjoined acts of accommodation and convergence—thus adding my testimony in the favor of iritic action in this most interesting case.

Dr. F. X. DERCUM.—The psychologists have been charged almost as a body with claiming that the pain sense and the tactile sense were the same thing. Now, clinically, we have all seen the separation of the tactile from the pain sense. If I understand the report of this case, this man shows the same thing. A patient at the Philadelphia Hospital with syringomyelia, has tactile sense with absolute loss of the pain sense over the right arm, shoulder, right side of neck and face.

Professor LIGHTNER WITMER.—I should say that psychologists as a body do not confuse the pain sense

and the tactile sense. There is a body of psychologists that consider pleasure and pain to be a quality of all sensation, not only of the tactile sense, but also of the color sense and the temperature sense. Mr. Marshall has presented one of the newest forms of this theory and I understand that certain of the neurologists of New York are rather tempted to subscribe to this theory, and that Dr. Dana, accepting the psychological analysis of Mr. Marshall and others, has come to the conclusion that it is useless for neurologists to hunt for pain nerves, because pleasure and pain are related through phenomena that are to be taken as different aspects of sensation; Mr. Marshall's peculiar theory being that they are due to nutritive conditions of the organ that may be involved. I, however, would not say that even a small body of psychologists subscribes to this view.

Dr. WHARTON SINKLER.—This case seems to me to be one of syringomyelia, although it is peculiar from the fact that the thermal anæsthesia is limited to one lateral half of the body and that there is not the dissociation of the pain and thermal sense with which we usually meet in syringomyelia. Of course, it must necessarily be that in this disease, there are different degrees of destruction in the centre of the spinal cord, and consequently varying symptoms. This case also differs from the most common cases of syringomyelia in the absence of muscular atrophy which is a common symptom in the absence of joint lesions and in the preservation of the muscular power for such a length of time. Nevertheless, I believe that it is perfectly proper to consider this a case of syringomyelia.

Dr. CHARLES K. MILLS.—One practical point in the diagnosis of this case seems to have been overlooked, and that is the question whether or not this man could have any localized unilateral lesion in his cervical spinal cord. I have seen two cases in which the diagnosis of syringomyelia was at least provisionally made; in one case the symptoms were loss of power and wasting in both upper extremities, with distinct changes in the pain and temperature sense and other symptoms which made up a fair picture of syringomyelia. This case came on after injury. He had, however, no deformity in the region of the neck or upper dorsal spine. While I discussed the question of syringomyelia with his physician, I also said that it was possible to explain the case by some lesion of the cervical spine. The man returned to his home, and

was treated by extension and similar measures and a year later was practically well. I know of another such case. The man before us to-night has no changes in the iritic reflexes, no loss of knee-jerk, no ataxic symptoms; he has an imperfect history of some pain lasting a short time. He has ankle clonus on both sides. The condition might be due to unilateral compression or partial compression of the cord, althoughit is not probable. This has an important bearing on treatment.

Dr. CHARLES S. POTTS.—In this connection I should like to mention a case which I saw at the University Hospital last summer a year ago. The patient had all of the classical symptoms of syringomyelia-atrophy of the muscles of the arms, spastic condition of both legs, increased knee-jerk, loss of temperature and pain sense and preservation of the sense of touch. I made a diagnosis of syringomyelia. The man died sometime afterward and Dr. Guiteras made a post-mortem and said that there was no central lesion of the cord, but that there was cervical pachymeningitis. This man had, however, never complained of any pain. Microscopic examination of the cord has as yet not been made.

Dr. WHARTON SINKLER.—There is no question that cervical pachymeningitis simulates syringomyelia more closely than does any other disease. One can hardly imagine a pachymeningitis of the cervical region which could be strictly unilateral. This affection is almost always associated with pain and rigidity in the neck muscles. There are also muscular wasting, but no changes in the temperature and pain sense.

Dr. SAMUEL WOLFE read a paper on

POLIO-ENCEPHALITIS SUPERIOR ACUTA, WITH REPORT OF A CASE. (See page 53.)

DISCUSSION.

Dr. CHARLES A. OLIVER.—I was much interested to hear Dr. Wolfe's account of the total exterior and interior ophthalmoplegia in his case. My own experience with such a type of paralysis has been limited as I have seen the onset and disappearance of the symptoms in but two cases of total involvement of the third, fourth and sixth nerves; both of these cases, however, were not of the exact type described to-night.

One was the case of a lad, fourteen or fifteen years, who, although when first seen was unable to converge to the median line any nearer than about thirty-five centimetres, yet was able to readily have either internal rectus muscle respond when the opposite externus was brought into combination with the internal muscle during conjugate lateral deviation at fixation objects as near in as eight and ten millimetres. In this peculiar way his series of phsiologic muscle-groupings failed to act one after another during a period of some three or four weeks. I gave large doses of iodide of potassium, one hundred and one hundred and fifty grains daily, and the action of the muscles gradually returned in curious physiological sequences. In this case the interior ophthalmoplegia persisted longer in the iris grouping than in the cilliary muscle. The second case I saw only during convalescence. It was syphilitic in type and made a rapid recovery by the administration of large doses of iodide of potassium, one hundred to two hundred grains daily. It also passed through the same peculiar physiological changes; that is, the action of any one muscle under certain impulses seemed to be lessened in work in certain ways more than whilst it was under other conditions.

I have recently had a most instructive case of the chronic type in a child eight or ten years of age, in regard to which Dr. S. Weir Mitchell kindly gave an opinion. The paralysis was limited to the exterior group of the occular muscle. There was incomplete ptosis and there were some facial disturbances. After a careful analysis and general condition of the symptoms, and a study of the personal and family histories, Dr. Mitchell was of the opinion that the case was one of hereditary syphilis.

I have had some seven or eight more or less complete cases of the chronic types. In almost every instance that I have had opportunity to study, I have found some slight degree of optic-nerve degeneration with diminution of the fields of vision. In all they were concentrically diminished in size, especially for green and red. In one or two and at various times in the same case, the field borders appeared indentated.

Dr. CHARLES K. MILLS.—This case recalls a certain clinical type, which, I think, has not been recorded in any accurate manner, although the cases must have been observed. In these cases the affection comes on sud-

denly, practically as an apoplexy, paralyzing either the third or sixth nerve with anæsthesia to pain and temperature on one side of the body. I have seen several such cases, and I have seen them in two forms, a severe and a milder type in which approximate recovery takes place. They are nearly always syphilitic cases. They recover to a certain extent, but rarely entirely. The ocular conditions disappear more promptly than the disorders of sensation which may remain permanently.

Dr. SAMUEL WOLFE.—As I said in the paper, I think that syphilis can be excluded in this case. The possibility that the influenza poison was the cause in this case is well worth consideration. We must admit that in syphilis and in various other diffused inflammations within the cranial cavity the nuclei of the eye muscles are occasionally involved, more often unilaterally than bilaterally, but so perfect a type of ophthalmoplegia with so little disturbance of consciousness and so little general disturbance, can, I think, only come from an inflammatory lesion and a lesion which in its selective action elects the motor cells of these nuclei and leaves alone the surrounding tissues. I believe that all of the other symptoms in this case can be explained on the theory of indirect action,—certain nutritive and circulatory changes which are present in the neighborhood of the inflammatory focus. I, therefore, repeat that I believe that we have here a distinct disease entitled to this name and that this case is a typical instance and that the ophthalmoplegia which occurs in the course of other diseases can be best excluded by a study of the cause and taking into account the bilateral character and the absence of other symptoms due to direct action.

Stated meeting, February 26th, 1894.

President, Dr. WHARTON SINKLER, in the chair.

Dr. WILLIAM TAYLOR read a paper on

TREPHINING FOR MIDDLE MENINGEAL HEMORRHAGE WITHOUT FRACTURE—AND REPORTED THREE CASES.

ABSTRACT.

He described briefly the anatony of the parts involved, the symptoms usually accompanying rupture of the artery and gave a short account of the surgical technique.

CASE I.—A man, aged 48 years, who had fallen down stairs and injured his head, but with no evidence of fracture of the skull. He was stunned by the fall, but soon regained consciousness and walked around the room for some time. He again became unconscious and in this state was brought to St. Agnes Hospital.

He had constant convulsive movements of the right side and paresis of the left side of the body, with paralysis of the left side of the face.

A button of bone was first removed from the right side of the skull and the anterior branch of the middle meningeal artery found to be uninjured. A similar button of bone was then removed from the left side of the skull and the ruptured anterior branch of the artery secured by ligation and packing with gauze after a large blood clot had been removed.

The patient died in two days. There was no fracture of the skull, but an apoplectic clot in the right hemisphere of the brain was discovered. This was the cause of the left-sided motor disturbances. It was now known for the first time that the patient had had an apoplectic seizure some six weeks before the injury just described.

CASE II.—A man, aged 50 years, admitted to St. Agnes Hospital under the care of Dr. W. W. Keen. He had fallen from a hay loft, striking upon the top of his head. When seen by Dr. Keen, about one hour after the accident, he was unconscious, the left pupil dilated, but there was no fracture of the skull. The skull was trephined and the anterior branch of the middle meningeal artery was seen to be ruptured at two points, and the posterior branch was likewise ruptured at two points.

The patient died very soon from the loss of blood and severity of the injury.

CASE III.—Was one of intracranial (cerebral) hemorrhage, involving fibres from the cuneus and producing hemianopsia.

The patient was a slender woman of 65, who had fallen down stairs, striking the back of her head.

She was unconscious at first, but the next day, although not able to manifest desires, she found a lighted candle with her eye when the candle was carried to the left of the middle line, but when the light was carried to the right of the middle line she would not follow it. There was no paralysis at first, but gradually in about thirty-six hours after the accident, she became comatose and did not move her extremities.

No operation was attempted, as she was dying when first seen by Drs. Keen and Taylor.

Post-mortem examination showed no fracture, but a cortical clot extending deeply so as to moisten the fibres for the cuneus. This clot was directly beneath the posterior branch of the middle meningeal artery, and was the point selected for operative interference had that been possible.

DISCUSSION.

Dr. W. W. KEEN.—There are a few points to which I should like to call attention in connection with these cases. First of all, it seems to me that the gravity of the injury, the ease of diagnosis in the large majority of cases and the success of treatment are not appreciated either in our text books or by the profession at large. I am very glad that Dr. Taylor has reported three fatal cases, because we want to know something with reference to results with and without operation. If none of these cases had been operated upon they would all without question have died. Operation gave the only possible chance of recovery. The difference in result be-

tween the non-operated cases and the operated cases has been studied by Wiesmann, who places the mortality of the first class at 89·1 per cent., and of the second class at 32·7 per cent.

The mechanism of the tearing of these vessels was well illustrated to me last Wednesday in a case where I operated for the removal of the Gasserian ganglion. After turning down the flap of bone and scalp, as I was about lifting the middle cerebral lobe with my finger, suddenly a violent hemorrhage took place from the posterior branch of the middle meningeal and I perceived that it was due to the fact that a large branch was given off to penetrate the bone and when my finger lifted the dura this was torn. I have no doubt that in the majority of cases where this lesion follows a blow it is due to this momentary separation of the dura from the bone tearing the branches in this manner. There is, I believe, only one instance on record of spontaneous rupture of the middle meningeal. This was a French case reported by Michaux (*Rev. de Chir.*, 1891, 376). The man was an absinthe drinker. He recovered after trephining.

In making the diagnosis, the first point of all and the one of greatest value, as Dr. Taylor has indicated, is the period of consciousness between two periods of unconsciousness—the primary stunning and the secondary coma. This period of consciousness, if it exists, which is not always the case, should always make us suspect rupture of the middle meningeal. The man reported by Dr. Taylor, who had fallen on his head in a stable, had four ruptures, two of the anterior and two of the posterior branch. There was absolutely no history of consciousness, and I have every reason to believe that there was no period of consciousness, because, when I saw him about two hours after the accident, the clot was so large that when I opened the skull it took nearly the entire length of my finger to reach the dura. The clot was probably quite as large as my fist.

As to the localizing symptoms, they fall into three categories. In the first place there are the motor symptoms, because, fortunately, this artery lies in the neighborhood of the motor zone. We have motor aphasia from pressure at the bottom, then involvement of the face, then of the arm, and rarely, unless the hemorrhage be enormous, involvement of the leg centres. I think we cannot insist too strongly that the site of all operations should be determined, not by the seat of the injury or

supposed seat of the injury, but by the localizing symptoms. A number of instances has been recorded by Jacobson and others of erroneous treatment owing to the fact that localizing symptoms were not depended upon.

We have, secondly, the dilatation of the pupil as another localizing sign. In one of the cases reported the dilatation of the pupil was the only localizing symptom that I had. The pupil on the left side was a little larger and a little more sluggish than that on the right and this determined me to operate on the left side. If this had failed, the only proper course would have been to have operated on the opposite side. In the last case narrated the very careful observation of Dr. Cross was the only localizing symptom. If we had made a diagnosis from the movements of the patient's hand to the right side of the head, we should have been wrong. The observation of Dr. Cross seemed to indicate that the left half of each retina was blind, and, therefore, the left optic tract and the left cuneus were involved.

With regard to the diagnosis between rupture of the middle meningeal and the middle cerebral artery, I do not think that a satisfactory and accurate diagnosis can be made, nor do I think that this is essential. Whether the clot be inside or outside of the dura is, surgically speaking, a matter of small moment.

The control of the hemorrhage should, if possible, always be by ligature. Sometimes, where there is rupture of the main trunk low down near to or at the foramen spinosum, this may not be possible. In these cases the hemorrhage can sometimes be prevented by packing. I had this illustrated last Wednesday when removing the Gasserian ganglion. In attempting to discover the third division of the fifth nerve, using Allis's blunt dissector, the middle meningeal was ruptured in the foramen spinosum. So long as the dissector remained in the foramen, hemorrhage was arrested. While controlling the bleeding in this way I removed the third branch and destroyed the ganglion. I then substituted my left forefinger for the dissector. Then I smeared over a piece of iodoform gauze with Horsley's putty and inserted this over the opening, and then packed tightly with gauze. This controlled the bleeding, and three days later I removed the gauze with great care and the hemorrhage remained arrested. No bleeding has occurred since.

Dr. ERNEST LA PLACE.—Dr. Keen has covered pretty

much all the points, and I can add little except the report of a case. In this case there was that interval of consciousness lasting half an hour to which reference has been made. This man was struck on the side of the head with a whip handle, but there was no fracture. Half an hour later the arm became paralyzed and he gradually lost consciousness. He was then brought to the hospital and I immediately trephined and removed the clot. The bleeding was controlled by packing the iodoform gauze as the artery could not be readily found.

I note in the paper that the author uses cat-gut to sew up the meninges. I have always been afraid of cat gut, owing to the ease with which infection takes place. I have always used the finest Chinese silk and have had no reason to regret it.

In this connection, I should like to mention a case reported by Smart in Sajous' Annual, of hemorrhage at the base of the brain as the result of a fall without fracture. There was left hemianopsia, slight motor aphasia, and general dullness without complete unconsciousness. The diagnosis was not clearly made out, but in as much as the symptoms were progressive, it was thought wise to trephine, and Dr. Chiene, of Edinburgh, trephined on a level with the zygomatic arch and removed from the under surface of the brain a large amount of blood. The exact source of the bleeding was not made out. In his remarks he advises the use of the trephine somewhere in the neighborhood of the suspected hemorrhage, even if the absolute diagnosis cannot be made out. Some two years ago, I insisted on that point and related a case of hemorrhage at the base of the brain the result of a wound produced by a fencing foil. This injured the cavernous sinus leading to the formation of a clot. I trephined in the same locality as Dr. Chiene and removed a large clot. The following day the dressings contained at least four ounces of serum. I believe that there is still a large field to be studied in the way of thorough drainage of the cranial cavity.

Dr. FRANCIS X. DERCUM.—In regard to supra-dural hemorrhage, the symptoms, it seems to me, are usually clear, but both in supra-dural hemorrhage and in intra-meningeal hemorrhage doubt now and then arises when the hemorrhage is complicated with signs of concussion. To illustrate this, I might recite a case which occurred in the Philadelphia Hospital some years ago. An epileptic fell in a fit striking the back of the head on the

hard pavement. One of the surgeons happened to be in the institution and I summoned him in consultation. The man was unconscious and had stertorous breathing, and it seemed that one side was a little more relaxed than the other, and on that side, also, the pupil was dilated. I suggested that the case be trephined. I did not expect to find supra-dural hemorrhage as the interval of consciousness was lacking, but thought that hemorrhage into the arachnoid space would be found. Although the symptoms seemed to point distinctly to one side, trephining on that side failed to reveal the clot. The patient's condition was such that the surgeon declined to go further. The autopsy showed an extensive subdural clot on the other side. Trephining could hardly have afforded relief as the effusion was much diffused over the lateral aspect and the base of the brain. Where the bleeding is so widely diffused, the mechanical difficulties might be overcome by making openings in the skull some distance apart and irrigating from one to the other.

In a case in which we have symptoms of cerebral concussion complicating those of fracture or hemorrhage, trephining should be done with great rapidity, and failing to find the source of trouble on one side we should then trephine on the other. The added number of openings in the skull will do no harm.

In some of these cases I do not see the way clear to make a differential diagnosis, and to say how far the symptoms are due to concussion and how far to cerebral hemorrhage, and I should like to hear an expression of opinion from the surgeons on this point.

Dr. WHARTON SINKLER.—I saw Mrs. A. with Dr. Cross, on the evening of December 30th. I found her comatose, breathing stertorous, and entirely unconscious of all her surroundings. The pupils of both eyes were widely dilated, but the coma was so deep that she did not observe light, even when a candle was held close to her eyes. There were involuntary movements of both the left and the right arms. These movements were not spasmodic, but the arm was lifted and moved about as if under the influence of the will—the right arm was used most freely. There was no evidence of any sensation to touch, or pin pricks. The knee-jerks were absent; and irritation of the soles of the feet caused no reflex contractions. A careful examination of the scalp showed no external evidences of injury, and no depression in the skull could be detected.

Taking the history of the case in consideration, and Dr. Cross's observation in regard to the presence of hemianopsia, it seemed that there had been a hemorrhage sub-dural or extra-dural in character, and in the region of the left cuneus. The absence of paralysis made it improbable that there had been an extensive hemorrhage in the substance of the brain as this would, no doubt, have reached the motor region. As the patient's pulse was good, the heart sound and the general condition fair, it seemed a case in which a trephining over the seat of the supposed lesion would be a justifiable operation. At all events, the prognosis was almost surely fatal if no operative interference was made, and, under these circumstances, the question of the operation with a full explanation of the condition of affairs was laid before the family. They at once agreed to take the chances of an operation, and I, therefore, asked Dr. Keen to visit the patient, with a view of trephining. The patient died shortly after Dr. Keen reached the house. The death was quite sudden, from failure of the heart. A few minutes before, the pulse was good and the patient's condition apparently as favorable as it had been three or four hours previous. The autopsy, of which Dr. W. J. Taylor has given a description, shows that had an operation been done, it would have been easy to remove the clot which was found at the autopsy.

The question, as to whether the hemorrhage had preceded the fall, or was the result of a blow upon the head, is one of great interest. The patient had a history of one or more attacks of vertigo previously, and it seems not improbable that she had an attack of vertigo while going up stairs and fell in consequence.

The diagnosis between sub-dural or extra-dural hemorrhage, and hemorrhage into the substance of the brain, is one which it is not always easy to make. Hemorrhage into the substance of the brain, however, usually occurs from the middle meningeal artery, or one of its branches, and the hemorrhage then takes place into the internal capsule, or in the motor region of the cortex. Under these circumstances there would, of necessity be paralysis of motion, more or less extensive, as well as compression symptoms. The history of the case is of great assistance in deciding the question as to whether the lesion is meningeal or cerebral. The operations which have been done for hemorrhage just beneath or over the dura have been few in number, but the results have been

sufficiently satisfactory to make us feel encouraged to operate in a certain class of cases. The cases most favorable are those in which an injury has caused the lesion and in patients whose vessels are healthy.

When we see a case like that which has just been referred to, in which localizing symptoms are so well marked, I think it is our duty to recommend a surgical operation, provided that the patient's condition is sufficiently good to justify it. The case under consideration was hardly a good subject for operation; her age was advanced, the vessels were atheromatus, and it was a question whether the hemorrhage was the result of the fall which she had sustained or whether a diseased vessel had given way; so that the operation would have been attended with difficulties, and it is not likely that even had the operation been successfully done, that the patient would have survived many months.

Dr. W. W. KEEN.—With reference to the diagnosis between concussion and hemorrhage, there are several symptoms which I think that we can utilize. The period of consciousness, if it exists, is of great value. In concussion there would not be that deep unconsciousness which we see in the secondary coma from pressure. In concussion the patient can usually be aroused by shaking or loud questioning; in the coma of compression this cannot be done. In concussion the pupils are usually equal and responsive to light. In compression from clot the pupils are usually unequal, and the one on the side of the pressure will be more or less dilated, and also be very sluggish or not at all responsive to light. The rise of temperature which follows middle meningeal hemorrhage is not usual in connection with concussion. As time goes on, if the symptoms are to concussion, the condition of the patient will improve, whereas if they are due to hemorrhage, they will go from bad to worse. Moreover, in concussion alone, there would not be distinct localizing symptoms.

I should like to emphasize the statement already made that if we do not find the clot on one side, we should trephine on the other side. Especially, as there are on record a few instances in which the localizing signs have been misleading and the hemorrhage has been on the side opposite to that indicated.

Dr. Taylor has confined his remarks to cases of hemorrhage without fracture. These, of course, are much fewer than those with fracture. I do not think

that we can too strongly impress the profession at large with the fact that with moderate violence, wholly insufficient to fracture the skull, the middle meningeal or middle cerebral may be ruptured, and, as a consequence, death will almost invariably result unless operative interference is undertaken.

Dr. F. X. DERCUM.—I alluded not so much to the differential diagnosis between concussion and meningeal hemorrhage, but to the difficulty in recognizing cases in which the concussion is a complicating factor.

Dr. CHARLES K. MILLS.—Several points in connection with localization are of interest. A case was mentioned in the paper in which there was spasm on one side and loss of power on the other; and the spasm was on the side opposite the lesion. Dr. Taylor also said that the hemorrhage was dural and made the statement that the symptoms would probably be the same as when the hemorrhage was subdural or cortical. This may or may not be the case. This is occasionally a source of error in studying localization symptoms. I called attention to the fact in 1888, which has been referred to before, that the spasmodic symptoms of dural hemorrhage are, or may be, on the same side as the lesion, and that this is probably attributable to the fact that the spasm is the result of conveyed or reflected irritation through branches of the fifth nerve to the pons. In these cases the spasm is likely to have a large tonic element. You may have this in sub-dural, but more especially in supra-dural clot. In some of these instances there may be an association of both dural and cortical spasms.

Another point which is passed over too frequently is the association with the fracture or large hemorrhage, of numerous small hemorrhages in various portions of the brain. The same thing occurs in a case of large hemorrhage into the brain without external violence. Duret has indicated the mechanism of the occurrence of these hemorrhages. I might say that these minute hemorrhages are particularly liable to occur when fracture has not taken place. When we come to the consideration of confusing symptoms it is important to bear in mind the possibility that the symptoms referable to the other hemisphere may be due to the more or less minute extravasations which accompany the large and supreme extravasation.

A symptom which has always interested me in these cases is the dilated pupil. I have never been able to

reach an entirely satisfactory explanation of the occurrence of this symptom. As I have observed these cases, it is not accompanied by other indications of third nerve paralysis. What would seem to be a simple explanation is, that the blood goes to the base and by direct pressure on the third nerve or some of its root fibres causes the symptom, but this does not seem to be likely. Various other explanations suggest themselves, but none are sufficient.

When one talks about differentiating concussion from hemorrhage it is necessary to know what is meant by concussion. As I understood Dr. Keen's remarks, he rather indicated what we know in popular language as a stun from a blow, which is often seen and from which the patient recovers. We have all varieties of stunning beside those in which the patient rapidly recovers without any after effects. In some varieties of concussion you can have more serious and fatal symptoms than occur in ordinary hemorrhages.

Dr. HOWARD F. HANSELL.—Since dilatation of the pupil has been referred to as a diagnostic symptom, I have been endeavoring to find in my own mind a satisfactory explanation of it. While I believe that it can help to indicate the side of the brain affected, I think that its localizing value ceases there. We know that dilatation may be due to paralysis of the pupillary branch of the third nerve, in which case the lesion will be nuclear and situated in the floor of the fourth ventricle, or it can be due to over-action of the sympathetic fibres without paralysis of the pupillary branch. But neither of these explanations is satisfactory, and I have concluded that the dilatation must be because the optic nerves on that side are paralyzed by pressure. The light reflex is a circle, the first part of which is the impression on the retina, which is carried back through the optic nerve. If the retina is not perceptive we will have dilatation of the pupil. In nearly all cases of blindness from paralysis of the optic nerve, we find a dilatated pupil, and this without any affection of the sympathetic or the third nerve.

The diagnosis of hemianopsia, present in one of the cases, must have been attended with considerable difficulty. There are central visual fibres in the optic nerve which run out from the fovea centralis of each retina, and these are seldom affected in hemianopsia. Since the image of an object looked at falls on the fovea centralis and its immediate neighborhood, the visual axes would

naturally be directed towards a light, even in cases of hemianopsia. The symptoms of dilated pupil and hemianopsia are, in my opinion, simply indications of the side of the brain on which the lesion is and do not localize definitely.

Dr. W. W. KEEN.—I would state that in the third case there was not dilatation of the pupil on one side, but both pupils were dilated. While I admit that Dr. Cross's observation as to hemianopsia was a crude one, yet it was necessarily so, and it was the only localizing point that we had. The autopsy showed that had we operated on the indication that it furnished, we should have been correct.

Dr. ERNEST LA PLACE.—With regard to the diagnosis between concussion and other conditions, I take it that concussion is really, so far as diagnosis is concerned, purely functional. It is a shaking up of the brain, it is a mild striking of the brain against the skull. This is the mildest type. Anything beyond that to the extent of the rupture of the minutest capillary ceases to be concussion, but enters the domain of contusion. The extent of the contusion may vary to any degree depending upon the number of capillaries that have ruptured. This causes hemorrhage and leads to compression. Where there is simple concussion the man is dazed for a time but he comes out of that and there is no permanent change. Where there is contusion there is hemorrhage, the effects of which will remain until absorption has taken place, and even after absorption, and may result in certain forms of insanity, of permanent headache or of epilepsy.

Dr. CHARLES K. MILLS.—In the sense in which Dr. La Place defines concussion, I am inclined to doubt if we ever have unconsciousness produced. Of course, theoretically, it is possible for the brain to be concussed without hemorrhage taking place. The blood vessels of the nervous system, and the nervous structures themselves, are so delicate that I do not accept molecular concussion as taught by Erichsen as explaining many phenomena. In all the severe cases of concussion it is probable that hemorrhage from the capillaries at least takes place. I do not believe that this is the result of contusion in the sense of impact against the inside of the skull. The physical principles underlying the occurrence of these hemorrhages have been explained by Duret and others, and the hemorrhages may occur in parts of the brain

not in contact with the skull. Concussion, when severe enough to produce unconsciousness is, I presume, always associated with some vascular lesion. With regard to the explanation of the dilated pupil, I do not think it necessary to bring hemianopsia into the discussion. The explanation is probably that which has been indicated, that the fibres which complete the reflex pupillary arc are interfered with. It has been shown by microscopic examination that one little set of fibres of the optic tract dip into the gray substance of the base of the brain near the tuber cinerum, and pressure on the optic tract of one side including these fibres might explain the dilatation of the pupil.

Dr. F. X. DERCUM.—I cannot accept the explanation of the dilatation of the pupil which has been given. The fibres of the optic tract which form part of the arc for the reflexes of the iris go to one half of each eye, and thus the retinæ of both eyes are equally deadened and there is slight dilatation of both pupils, never inequality.

Another explanation is this: The nuclei of the oculomotor nerves, which give origin to the fibres supplying the iris, are exceedingly delicate. They are much less robust than the cells which supply the gross muscles, and we know further that these fibres react to very delicate and comparatively slight impressions; thus, in syphilitic cases we often see marked dilatation when there is only slight involvement of the extrinsic muscles. In hemorrhage, we have a sudden increase of the intracranial pressure and these delicate structures react readily to slight changes in pressure, and that is why we have the dilated pupils.

Dr. W. W. KEEN.—The word "concussion" when used symptomatically is a very convenient one, but when used pathologically it is, I think, inaccurate. I do not believe in concussion as expressive of simply a functional lesion. I cannot conceive of there being symptoms of concussion of any severity without some physical change in the brain itself. This change may be a rupture of the capillaries, but we must remember also that the nerve fibres are quite as delicate and as easily ruptured as the capillaries. I, therefore, believe that the word concussion, in a pathological sense, indicates a lesion, it may be of the blood vessels or of nerve fibres or possibly of the nerve cells, or of all these structures.

Dr. W. L. ROBINSON, Danville, Va. (By invitation).—I am not a neurologist, and in the line of thought that

you have been discussing, I do not think that I could add anything. If, however, two cases that have occurred to me in the last eighteen months would be of interest to you, I shall gladly relate them. Eighteen months ago, I was called to see a little child, three years of age, who had fallen some twenty feet, striking the head on the edge of a plank. There was a long swelling half as thick as my wrist on the right side of the head. The strange thing was that there was paralysis of that side. There was great stertor and difficulty of breathing. The skin was not broken. I had no instruments with me, so I sterilized my pen-knife and some gauze and opened the scalp and turned out the clot. Instead of finding a depression of the bone there was a marked separation; it seemed to be an eighth of an inch wide by five inches in length. After removing the clots, I laid a piece of gauze over the wound and gave an injection of chloral and kept the child thoroughly narcotized. The moment that it came from under the chloral, spasm would commence on that side. The child was seen by a number of physicians and they all said that it would die. I kept it under chloral three days and nights. I then let up on the chloral and it recognized its mother and gradually recovered without spasm. Twelve months later, I again saw it, a healthy, strong child without any paralysis.

The second case was that of a young man who, on the 12th of July, was diving in shallow water and struck his head and did not come up. His friends pulled him up. His family physician was with him at the time and thought that there was nothing wrong. Four men brought him to the hotel. He had no power of motion on either side below the neck, but was perfectly conscious. I made his comrades hold his heels, while I made traction on the head and got sensation in the fingers. I then applied extension and counter-extension and kept him so. I subsequently put on a plaster cast. In November, his physician removed the cast and he was a well man. You gentlemen can explain the case, I cannot, except by a dislocation of the vertebra.

Dr. CHARLES S. POTTS read a paper on

A CASE OF ACUTE DELIRIUM, WITH AUTOPSY AND BACTERIOLOGICAL EXAMINATION OF THE CEREBRO-SPINAL FLUID.

ABSTRACT.

The patient, a member of the medical profession,

æt. 33 years, of good habits, but sensitive nature, had been having considerable family trouble and worry. In June last was afflicted with urticaria, lasting until August, when a carbuncle made its appearance upon his upper lip. This was opened and curetted ten days after its appearance. For several days previous to its being opened the patient had been taking rather large quantities of whisky and morphia. This was stopped when he came under medical supervision, and he did well for nine days, when at 10:30 P. M. he became violently delirious and endeavored to jump out of the window. The next day he was, with the exception of brief intervals, rational, but in the afternoon again became violently delirious, and remained so until death. His temperature was normal until the evening of August 24th, three days after the commencement of the attack, when it was 101° Fahr. From that point it gradually arose until three hours before death, when it was 108·2° Fahr. At that time delirium gave place to coma and the patient died at 11 P. M. At no time was headache complained of. The autopsy was made seventeen hours after death by Dr. J. W. McConnell in the presence of Drs. C. K. Mills, Berlet, Klemm and myself. The brain and meninges appeared to be normal, as did all the other organs of the body.

Cultures made from the cerebro-spinal fluid by Dr. D. Braden Kyle demonstrating the presence of the so-called micrococcus lanciolaturs or pneumonococcus of Fraenkel and Weichselbaum, and the staphylococcus pyogenes aureus and albus.

Microscopic examination of the gray matter of the cortex showed the appearances described by Osler, *i. e.*, perivascular exudation, and leucocytes in the lymph sheaths and perigangliar spaces.

As the germs isolated in this case are those usually found in meningitis we would advance as a possible explanation for the lack of microscopic findings in spite of the severe symptoms present in this disease, that the taxæmia was so violent as to cause the death of the patient before appearances visible to the naked eye had time to develop.

DISCUSSION.

Dr. CHARLES K. MILLS.—I saw the case in consultation, but have nothing especial to add. The case presented every appearance of acute delirium. It is to be

regretted that special precautions could not have been taken in making the autopsy, but at the same time I believe that the observations are of value.

Dr. F. X. DERCUM.—This report is interesting on account of the discovery of the pneumonococcus. We are told by German writers that the pneumonococcus is by far the most common germ found in certain forms of meningitis, especially certain purulent forms though it is usually associated with other forms of pyogenic bacteria. Its discovery in a case, the history of which corresponds with that of acute delirium, is interesting. We have on other occasions discussed the connection between acute delirium and meningitis, and I think that the concensus of opinion was that we had infection in many cases of acute delirium. In this case it looks as though there would have been purulent meningitis, had the patient lived long enough.

Dr. ERNEST LA PLACE.—I should like to add that the pneumonococcus of Fraenpel inhabits the mouth and is very common. We now think that pneumonia is due to the infection of the lung with a germ that is often present in the mouth. Although many examinations of the blood might not reveal the germ, yet now and then we find germs in the capillaries. If we puncture the finger and examine the blood, it is possible that we will find nothing. Even if in two or three hundred examinations we find nothing, we must remember that we have examined only a few drops of blood. There may be germs present, and if only one travels to the spot, that one is sufficient to start the infection.

TRAUMATIC AFFECTIONS OF THE CERVICAL REGION OF THE SPINAL CORD, SIMULATING SYRINGOMYELIA.

By JAMES HENDRIE LLOYD, A.M., M.D.,

Physician to the Philadelphia Hospital.

THE earlier writers, of what may be called the present epoch of clinical neurology, recognized and described a particular type of lesion of the spinal cord which was localized in the cervical enlargement and the pathological process underlying which was supposed to be always inflammatory. With his keen instinct for novel clinical types, Charcot[1] was among the first to recognize this affection, to give it an appropriate name, and to write an accurate description of it founded upon an autopsy. He called it *pachymeningitis cervicalis hypertrophica*, a name which indicates sufficiently both its nature and its seat. He was assisted in some of these observations by Joffroy, who subsequently wrote his thesis on this subject.[2] It is but just, however, to say that before the time of these observers Abercrombie and then Gull,[3] in England, had put on record similar cases,

[1] Arch. de Physiologie, 1869 (with Joffroy).

[2] De la Pachymeningite Cervicale Hypertrophique. Paris, 1873.

[3] Guy's Hospital Reports.

but had failed to draw attention so accurately to their nature. Vulpian, subsequently, in his treatise on diseases of the nervous system, gave a concise description of this cervical affection, and later still, Burtin [4] wrote his thesis on spinal pachymeningitis, in which he described two types: first, the cervical, second, the lumbar. This cervical type of cord-lesion has become so well recognized now that most of the text-books describe it, although there are still a few that do not appear to recognize this affection, which can scarcely be called uncommon, and which presents some problems both in physiology and in pathology which give it quite an unique importance. These problems relate, first, to the physiology of the conduction of sensation; second, to the differential diagnosis between several pathological states, each of which while distinct in itself, can produce a complexus of symptoms very similar to that produced by each of the others.

The symptoms of pachymeningitis cervicalis may be divided into two groups. (1) Those pertaining to the level of the cervical region, and (2) those which are produced by ascending and descending degenerations. Among the first are neuralgic pains in the occiput, neck, shoulders and arms; paralysis, with muscular atrophy, in the shoulders, arms and hands; and stiffness of the neck with deformity of the spine. Among the second are spastic paraplegia, with increased knee-jerks and clonus, without muscular atrophy in the legs, and with or without (often without) paralysis of the bladder and rectum. There are in addition various alterations of sensation both at the level of and below the cervical lesion. These alterations were not duly studied in the earlier cases; in fact, in most of the papers no mention is made of alteration of the various modes of sensation. One of Charcot's [5] early papers is an exception, however, for in it there is express mention of thermo-anæsthesia as a symptom in one of his cases. It now seems certain that

[4] De la Pachymeningite Spinale. Paris, 1878.

[5] Charcot and Joffroy. Op. cit.

in the majority of these cases there is some alteration of sensation, and that this alteration may be the "dissociation symptom" described as peculiar to syringomyelia; *i. e.*, thermo-anæsthesia and analgesia without impairment necessarily of tactile sense, although this latter is occasionally modified in various areas. Other modifications, and combinations of modifications, of sensation will probably be found. Finally, there are sometimes trophic lesions in the bones and in the skin and nails, and there may be alterations in the pupils.

The pathology of pachymeningitis is familiar to those who have had opportunity to study lesions of the spinal cord.[6] As Vulpian describes this form of it, the cord is enlarged, filling the whole spinal canal in the cervical region. The enlargement is fusiform, and resistent on pressure, contrasting with the fluctuating sensation given by the cord in syringomyelia. The dura is much thickened and adherent, and the membranes separate from the cord with difficulty. The cord is sometimes fibrous, or it may be softened, and the gray and white matter cannot be distinguished easily. It contains no adventitious cavity as in syringomyelia. The nerve-roots are usually involved. As described by Vulpian and by Charcot in their own cases the appearances were not unlike those seen in tubercular pachymeningitis in the mid-dorsal region.

In presenting the brief histories of two patients this evening, I wish to emphasize the fact that both of them are traumatic cases, and both in men past the prime of life who had previously been in good health. It has been rather too much the custom in the past to regard cold as the most puissant agent in the production of all forms of pachymeningitis. That pre-eminence will probably be held henceforth by infection and by trauma. In presenting these cases, moreover, I purposely have called them simply examples of traumatic affections of the cervical cord, and I do not intend to commit myself to a

[6] See a paper, with illustrations, on "Pachymeningitis and Myelitis from Pott's Disease, etc.," by the writer, *University Medical Magazine*, Dec., '93.

statement that they are necessarily, or even probably, examples of an inflammatory process. They simply conform to a clinical type, due, in their cases, to trauma, and I confess that I am not satisfied in my own mind that they are not degenerative rather than inflammatory.

James G., aged 55, white, male, stonecutter by trade; gives a negative family history. He has no personal history of syphilis or alcoholism.

In 1876, he fell from a scaffolding in front of the U. S. Mint in this city, striking the back of his neck. A fracture of the spinal column was diagnosticated. The entire body, including both arms and legs, was paralyzed; breathing was embarrassed, and urination, but not defecation, was affected. It is reported that there was no marked disturbance of sensation below the waist, but the arms were anæsthetic. In three months he regained power in the legs; the arms did not improve so quickly, the return of power in them being delayed for eight months. He was weak for over two years; the neck was deformed; the right leg a little feebler than the left. In three years he returned to his work as a stonemason, claiming now that he was as strong then and as able to do hard work as ever. This return to work is a very significant incident in the history of the case. In 1884, five years later, he fell again, this time from a ladder, a distance of fifteen feet, and struck the back of his head and his spine between the shoulders. He was unconscious for six hours. This fall was followed by a return of the paralysis of the legs and arms, loss of control over the bladder, and obstinate constipation. According to his statement there was no marked loss of sensation, but we do not know how carefully that was determined. There was no priapism. There was severe pain at the seat of injury, and headache for three months. Power returned to the legs in two months, in the arms slightly at the end of seven months. After confinement to bed for four months, the patient began to get about, but he has not been able to do any work since the second accident.

The present condition is as follows: The patient has advanced atrophy of the shoulder-muscles of both sides, including the deltoid, pectoralis major, infra and supraspinati and the lower part of the trapezius; also of both the arm and forearm groups of muscles, and of almost

FIG. 1. A Case of Trauma of the Cervical Region of the Spinal Cord.

all the muscles of the hands. (See photograph, Fig. 1.) The thumb is in the position of extension of the metacarpal bone, flexion of the proximal and extension of the distal phalanx—not exactly the position assumed in "main-en-griffe," which position of the hand is not present here, although what seems to be a commencement of it is seen in the middle and ring fingers. Fibrillary contractions are well marked in some of the wasted muscles. The myotatic irritability is much increased, especially in the biceps and in the extensors of the wrist. There is a little power of flexion and extension of the forearm, but except for this, the arms are quite paralyzed and useless. The muscles respond to both currents; there is some decrease in the most wasted muscles, and some hyperexcitability, with duration tetany, in others.

The patient has not head-drop.

The legs are paretic and slightly contractured. The knee-jerks and ankle-clonus are very lively. There is no muscular atrophy in the legs. There are no trophic lesions, no sympathetic lesions, no bulbar symptoms, no disturbance of respiration, nor any paralysis of the bladder. There is marked stiffness of the neck, with deformity of the cervical spine. This deformity is a hump-like deviation of the spine, such as may have been caused by a fracture. There is also slight scoliosis. There is still more or less neuralgic pain about the neck and the occiput.

The sensory tests in this case are of great interest. (Figs. 2 and 3.)

Tactile sensation is good everywhere; repeated tests have always demonstrated this.

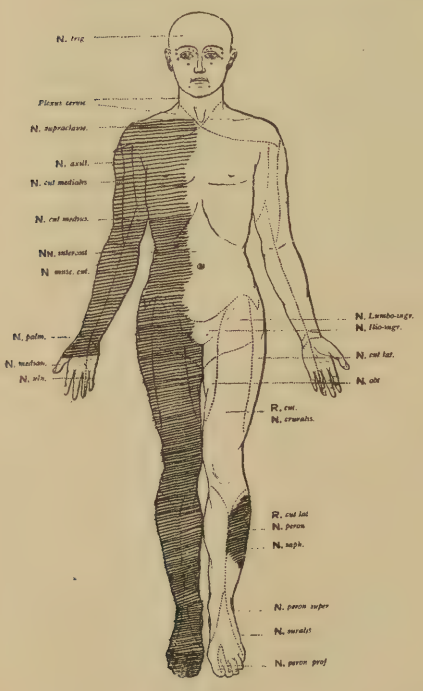


FIG. 2.

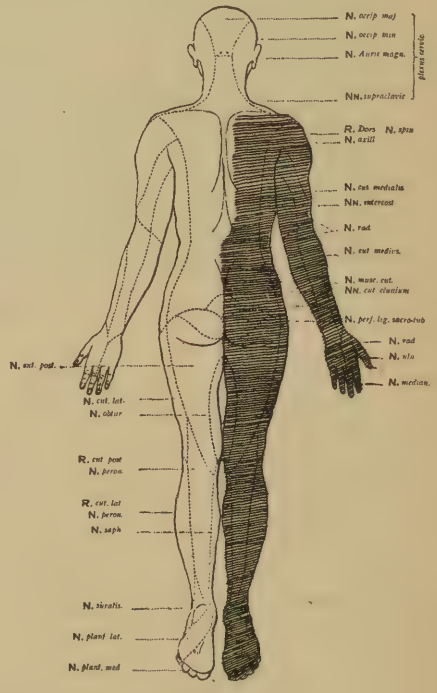


FIG. 3.

FIG. 2. Thermo-Anæsthesia and Analgesia on right side. Tactile Anæsthesia in small area on left leg.

FIG. 3. Thermo-Anæsthesia and Analgesia on right side. Tactile Anæsthesia in small area on left leg.

Thermo-anæsthesia is marked on the right side, as shown in the schemata. It is more properly an anæsthesia to cold. The patient distinguishes heat in the affected areas, but everywhere in these areas calls cold hot. There is analgesia quite marked in some of the affected areas. This condition has practically remained unchanged for a year, very recent tests corresponding closely to those made one year ago.

It is thus seen that this patient presents a very close resemblance to a case of syringomyelia in the cervical enlargement. The motor and sensory lesions, their location and distribution, are closely analogous. Trophic and vasomotor lesions are alone wanting. The question, indeed, presents itself, whether the lesion is a true syringomyelia of traumatic origin. Bruhl,[7] in his monograph, recognizes trauma as a cause of syringomyelia, and several of his reported cases illustrate this etiology. One of Charcot's latest lectures [8] was on a case of cervical trauma, caused by a gun-shot, simulating syringomyelia. The injury was probably to one of the nerve-roots, and thermo-anæsthesia was a marked symptom. In our case, an autopsy alone, it seems to me, could determine the exact pathology.

The second case is briefly as follows:

Patrick C., aged 60 years; white, male, laborer; gives a negative family history. No syphilis. Thirty years ago, he was injured in the cervical and dorsal spine, by falling into a canal and being jammed between two canal boats. For treatment he was kept strapped to a long dorsal splint or platform for five weeks. In about three months he had so far recovered, that he returned to work, and continued to do hard work as a common laborer up to the time of his second accident. The second accident happened one year before his admission to the hospital, about three years ago. He was in good health at the time. While working with others digging out a clay-bank, part of the bank fell and buried him. He was under the slide for about one-half hour before he was rescued. He was unconscious and remained so for

[7] Contribution a l'étude de la Syringomyélie. Paris, 1890.

[8] Clinique des Maladies du Système Nerveux, Paris, 1892, p. 333.

twenty-four hours. On recovering consciousness, he was found to be paralyzed in the left arm and leg. The face was not involved, and there was no affection of speech. He was in the hospital for the accident about six months. Contractures gradually began in the affected arm and leg.

FIG. 4. A Case of Trauma of the Cervical region of the Spinal Cord.

His present condition is as follows: He has a marked angular deformity of the first dorsal spine, and less markedly of the second dorsal and of the seventh cervical. There is a depression also in the region of the lumbar vertebræ. The patient carries his head in a set position due to the stiffness and deformity in his neck; his chin almost rests upon his thorax (see photograph Fig. 4). There is paralysis decidedly more marked in the left arm and leg than in the right, with contracture of the hand and forearm. The shoulder-muscles, especially the infra and supra-spinati, are atrophied, as are also the

muscles of the arm ; and there are fibrillary contractions in them. Clonus is marked in the fingers, wrist, and ankle of both sides. The phenomena of so-called "spinal epilepsy" are readily excited in the legs. This general leg-clonus, once started, will continue almost indefinitely.

The patient is unable to distinguish heat from cold, or to recognize either in the region marked on the chart. It will be observed that this thermo-anæsthesia is on the right side, or the side opposite the paralysis of motion. (Figs. 5 and 6.) There is an area of anæsthesia involving the right leg. (Fig. 7.) The man complains much, however, of a form of hyperæsthesia or pain, and paræsthesia of the right, or unparalyzed, side. He experiences constantly a sensation of burning on this side, and is in

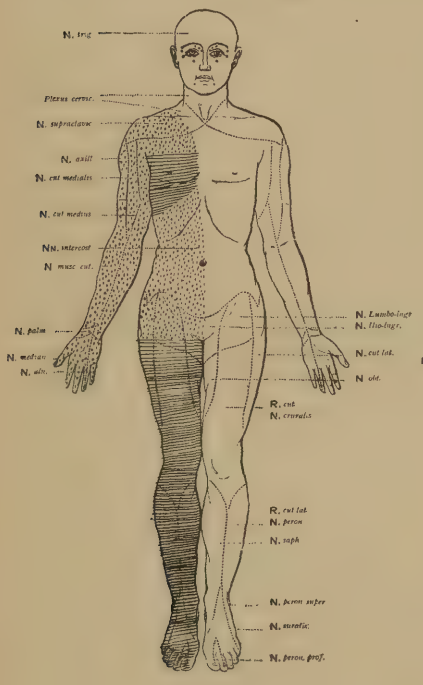


FIG. 5.

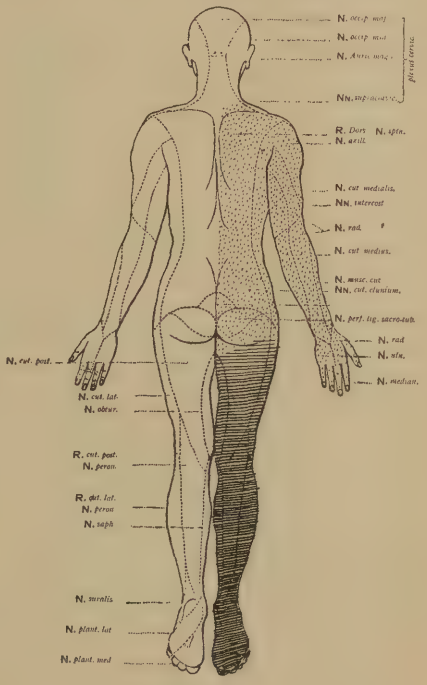


FIG. 6.

FIG. 5. Thermo-Anæsthesia indicated by cross-lines. Analgesia in the same areas, and also in areas indicated by dotted shading.

FIG. 6. Thermo-Anæsthesia indicated by cross-lines. Analgesia in the same areas, and also in areas indicated by dotted shading.

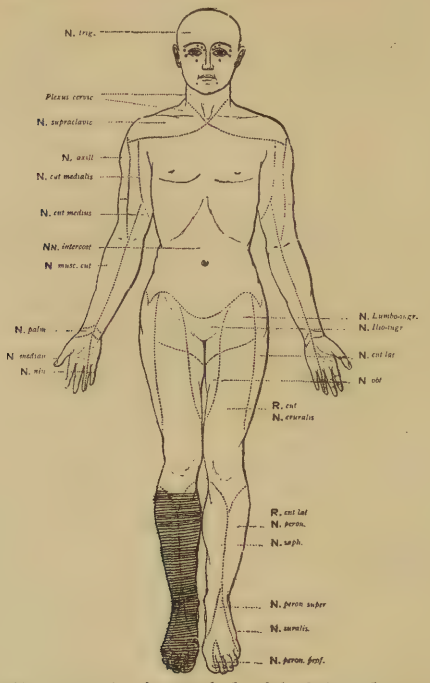


FIG. 7. Tactile Anæsthesia, varied with delayed sensation.

the habit of putting cold water on it to allay the heat, but he finds this remedy ineffectual—an interesting fact when it is recalled that he cannot distinguish cold from heat.

He is profoundly analgesic in the right arm and leg.

There is no involvement of the iris; no impairment of respiration, nor any interference with the innervation of the bladder.

It will be observed that the prominent feature in this case is its hemiplegic type. This is so pronounced that a hasty observer might be inclined to consider the case one of cerebral lesion. This view is opposed, however, by the history of the case, the direct effect of accident, the deformity of the spine, the absence of paralyses of the face or tongue, the sensory changes, and the atrophy and fibrillation in the paralyzed muscles. It follows closely the Brown-Sequard type—the paralysis of motion being on one side, the thermo-anæsthesia on the other. Its resemblance to syringomyelia is not so striking as that of the first case, yet it must be borne in mind that

there is an hemiplegic type of that disease. I once saw in my service at the Philadelphia Hospital a strict hemiplegia, the result of a lesion in the cervical cord, and some years ago, with Dr. Deaver, reported the case, in which the latter performed a laminectomy.

THE PHYSIOLOGY OF THE CONDUCTION OF SENSATION.

The hopeless confusion that has prevailed among physiologists upon the subject of the sensory paths in the spinal cord is known to all students. Their opinions are diametrically opposed to each other, and most of them are being contradicted by more recent and improved research. Among these opinions are the following[9]: That sensory impulses are transmitted by the lateral column of the opposite side; that they are transmitted by the lateral columns of the same side; that they are transmitted by the posterior columns of both sides; and, finally, that they are transmitted by the gray matter. Almost all of these statements are probably erroneous, unless amplified or modified as the case may be. The dictum of Brown-Sequard especially, that a hemi-section of the cord causes anæsthesia below the lesion on the opposite side of the body, a dictum that has been received unreservedly by neurologists for years past, must be regarded henceforth with grave suspicion. The causes for uncertainty and confusion have been of three kinds; the first has been the inability to control the secondary results of wounds before the days of aseptic surgery; the second has been the reliance placed entirely upon sensory responses in animals; the third has been the defect in histological study. All of these causes have either been removed or controlled to some extent. The methods of aseptic surgery have removed at least one element that formerly confused and even vitiated the results of experimenters.

The exact knowledge which we possess on the subject of sensory conduction in the cord is still limited, and when kept free from speculation and hypothesis

[9] Quoted from Gotch. "Recent Research on the Spinal Cord." *Liverpool Medico-Chirurgical Journal*, vol. xiii. 1893.

may be summarized very briefly. It is derived from three sources: (1) From histology. (2) From experiment. (3) From disease.

Histology.—According to Ramon y. Cahal, the sensory nerve-fibre, entering by the posterior root from its cell of origin in the spinal ganglion, divides dichotomously (or in half) in the postero-external column, one branch passing upwards in the postero-internal column, the other downwards. From these longitudinal branches there arise short transverse branches, which penetrate the gray matter, and there end in the tufts or "arborisations," which characterize the peripheral or distributory ends of nerve-filaments. This course is proved further by the fact that if a posterior nerve root is cut the ascending degeneration is seen only in the posterior column of the same side (Gotch). It is promptly checked in the gray matter, where the terminal arborisations occur, and no ascending degeneration is seen anywhere on the opposite side (Gotch). The ascending degeneration ceases after travelling up the posterio-internal column in the nuclei of the funiculus gracilis and funiculus cuneatus, which are ganglionic bodies, apparently beginning another stage of the sensory pathway. This view of the histology of the entering sensory nerve-fibre is adopted by Piersol, by Stirling and by Köllicker. In its simplicity it is very striking, and it leaves little room for the hypothesis that there are separate strands for tactile, thermal and painful impressions, at least in the posterior root. Certainly these impressions all enter the cord over identical fibres. It is lacking, absolutely, as far as it goes, in any proof of a sensory decussation low down in the cord. These tufts or arborisations, in which the sensory fibre ends in the gray matter, are apparently in close contact, but not in direct anatomical relation, with the ganglion cells, both in the posterior and anterior horns and in Clark's column, and from these ganglion-cells other nerve fibres are projected, the course of which, except those from Clark's column, which pass over to form the ascending cerebellar tract,

and those which pass out the anterior roots, is not very clear. Some of them pass up the antero-lateral columns, others decussate through the gray commissure with the elements of the opposite side; but the evidence from histology seems to be that these decussating fibres are relatively few, *i. e.*, in very small numerical proportion to that of the others. The contrast with the decussating fibres from the anterior horns, which run so conspicuously through the white commissure in front, is very striking.[10] Finally, it may be borne in mind that there is a very rich decussation of sensory fibres in the decussation of the fillet in the medulla oblongata, and that these are in direct line from the fibres in the posterior columns of the cord to the brain above.

Experiment.—Gotch [11] in his recent paper on the spinal cord makes the explicit statement that "recent physiological research shows that, in opposition to the views formerly advocated by many neurologists, the path for sensory conduction is almost entirely on the same side as that of the entering sensory nerves." Mott showed that the monkey after hemisection of his cord would remove a clip from the skin of the leg opposite the lesion, but would allow the one on the leg on the side of the lesion to remain. In the experiments of Gotch and Horsley the sensory tests, which are always uncertain in animals, were dispensed with, and the capillary electrometer was used instead. This instrument registers the passage of a nerve-current, and in their experiments seemed to show that mild stimuli send impulses up the posterior columns of the same side. These are the paths of least resistence. These mild stimuli represent probably simple tactile impressions, passing up the most direct way; *i. e.*, by the posterior columns. More violent

[10] If the evidence from histology is to be accepted there is as much, or even more, reason to say that the motor fibres decussate at each level of the cord as there is to claim that the sensory fibres so decussate. The truth probably is that all these fibres, in both the anterior and posterior commissures, are purely commissural.

[11] *Op. cit.*

impressions, however, such as would be made by heat, cold and pain, radiate through into the gray matter of the cord. Here they have a path of greater resistance (as we have seen histologically, a path not of direct continuity, but through the tufts of fibrils of the sensory fibres, hence passing in some way to the cells of the gray matter), and through the gray matter they possibly emerge again into the white conducting tracts, even of the lateral, and possibly of the opposite, posterior column. If accurate, this would mean that pain and painful temperature sensations are only of different degree, and hence excite a wider and more complex nervous mechanism than simple touch. From the clinical standpoint, the fact of separate alterations of the various modes of sensation by different diseases might thus be explained.

Disease.—In this connection I desire simply to call attention briefly to the fact that not only diseases involving extensively the gray matter, as syringomyelia, cause alterations in the temperature sense, but also that diseases of peripheral parts, as pachymeningitis and neuritis, may also cause them. The patients whose cases I report to-night are possibly mixed cases of injury both to the cord and to the nerve-roots.

Stated Meeting, March 26th, 1894.

Vice-president, Dr. JAMES HENDRIE LLOYD, in the chair.

Dr. M. ALLEN STARR, of New York, read a paper on

LOCAL ANÆSTHESIA AND OTHER SYMPTOMS PRODUCED BY LESIONS OF THE CERVICAL CORD.

ABSTRACT.

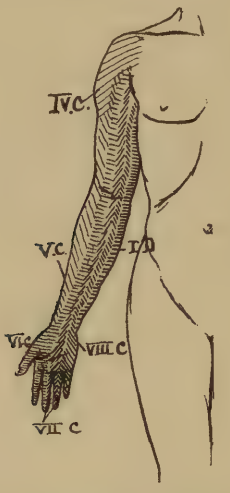
AFTER alluding to the relations between the various segments of the cervical cord and the spines and bodies of the cervical vertebræ, and showing that the relation is a variable one, the author proceeded to cite a number of cases of limited lesion of the cervical cord, traumatic, hemorrhagic, inflammatory and due to tumor, in which the local symptoms had been exactly studied, and in which the anæsthesia present had been carefully laid down on diagrams.

He then drew the following conclusions:

CONCLUSIONS.

First. As to the distribution of anæsthesia in lesions of the cervical enlargement of the spinal cord. By combining the diagrams already given with numerous other diagrams published by Thorburn and others, and comparing the results with descriptions of numerous cases

collected by Mills and Lloyd, by Chipault, White and other surgeons, it is possible to produce a schematic dia-



gram, such as is here presented, demonstrating the areas of skin upon the arm which are related to the various segments of the cord between the second dorsal and the fourth cervical.

1. The first area is a narrow one upon the inner side of the arm and forearm, extending down to the wrist, but not involving the hand. This includes a small zone both upon the dorsal and palmar surfaces of the arm and forearm, but is quite narrow. While it is probable that the upper portion of this area in the axilla is related to the second dorsal nerve, as Head maintains, I can find no exact lesion of the cord limited to the second dorsal segment and associated with such a limited anæsthesia to prove this statement.

There is a case reported by Charcot,[1] in which a bullet wound involved the first dorsal nerve only, and in which a small area in the axilla remained sensitive; the other parts included in area number one being anæsthetic, but as no examination of the cord was made in this case the conclusion drawn from it is of no more weight than that which might be drawn from a knowledge of the anatomy of the intercostal-humeral nerve. Area number one corresponds to the first dorsal segment.

2. The second area includes the ulnar side of the

[1] Arch. de Neurol., 1891.

hand, both palmar and dorsal surfaces, the little finger and one-half of the ring finger. It also includes a narrow strip of the skin upon the dorsal and palmar surfaces of the forearm and arm up to the axilla. This is proven by the fact that when lesions invade the eighth cervical segment of the cord the zone of anæsthesia upon the front and back of the arm and forearm is decidedly wider than when the first area alone is anæsthetic. It affords also a confirmation of the statements of Sherrington that there are no isolated patches of skin entirely separated from the body related to the sensory nerves. The second zone corresponds to the eighth cervical segment.

3. The third zone extends to the middle line of the middle finger, includes the middle of the palm and back of the hand and runs up the centre of the forearm and arm on both surfaces. This corresponds to the seventh cervical segment.

4. The fourth zone includes the remaining skin of the fingers and thumb and of the hand as high as the wrist and a narrow strip of skin up the forearm and arm on both surfaces to the axilla. This corresponds to the sixth cervical segment.

5. The fifth zone includes the skin upon the outer side of the forearm and arm on both surfaces as high as the insertion of the deltoid muscle. This corresponds to the fifth cervical segment.

6. The facts that in lesions as high as the fifth segment there occasionally occurs hyperæsthesia over the top of the shoulder and in the region of the supraclavicular nerves, and that in pachymeningitis the pain is felt here, leads to the belief that the fourth segment is in relation with this area. But in as much as lesions of the fourth segment of a destructive nature are uniformly fatal no case of anæsthesia can be found to determine the exact extent of skin which corresponds respectively to the fourth and third cervical segments.

It is hardly necessary to point out the differences between the areas of anæsthesia produced by cord lesions and those produced by lesions of individual nerve trunks, although a glance at the diagram and its comparison with Flower's well known plates shows this immediately. It is also evident that a study of the anæsthesia occurring in spinal cord lesions and that occurring in multiple neuritis, affords an important point of diagnosis between these two conditions. It will be remembered that in multiple neuritis, the anæsthesia affects all the fingers

simultaneously, then the entire hand and then extends up the arm involving all surfaces on both sides in an area which has been aptly described as the "glove area," corresponding, as it does, to the region of skin covered by a glove of various lengths. I have never seen a case of multiple neuritis in which the anæsthesia extended as high as into the axilla. It is usually limited by a line drawn around the elbow or a line in the middle of the arm.

Lastly, the area of anæsthesia occurring in hysterical conditions is different from that described as occurring in spinal lesions, for there is never the patch of normal sensitive skin over the top of the shoulder and down the outer side of the arm to the insertion of the deltoid, such as we have seen in all the cases of spinal lesions, and in hysterical anæsthesia it is well known that the neck is usually anæsthetic.

Clinical observers in the future should attempt by the aid of diagrams to define a little more exactly the region of the anæsthesia in the arm, and should also seek for a narrow zone of hyperæsthesia bordering the normal skin and between it and the insensitive region, which zone will necessarily be very narrow in the upper extremity. Herter is the only one who has described such a zone. In discussing the anæsthesia from lumbar lesions, I was able to show that such a zone of hyperæsthesia often existed of a most irregular shape in unexpected areas, which were easily explained when the exact region of the skin, corresponding to the segment just above the lesion was determined.

Secondly. Lesions in the lower cervical region of the cord produce a number of symptoms due to a lesion of the cilio-spinal centre which have been frequently overlooked by observers. The situation of this centre is in the first dorsal segment of the cord, and according to Klumpe[2] the symptoms are only produced by a lesion of the cord or of its nerve roots not by a lesion of the nerves after they have issued from the spinal canal and formed the brachial plexus.

The symptoms alluded to are myosis with sluggish reaction of the pupil both to light and in accommodation, a decided narrowing of the palpebral fissure so that the eye-ball looks smaller than normal and a slight retraction of the eye-ball. There is no flattening of the face, nor are there any vasomotor symptoms such as may be

[2] Klumpe. Paralysis Radiculaires. *Rev. de Med.*, 1885, Nos. 7 and 9.

produced by a lesion in the course of the cervical sympathetic nerve. These symptoms are very easily overlooked if they are present upon both sides. But when the lesion has been unilateral they have been noted by a number of observers.

Kraus [3] has described them in a number of cases of traumatic injury of the cervical cord due to fracture of the seventh cervical vertebra.

In a case of unilateral compression of the cord by a fracture of the seventh vertebra which I recently saw with Dr. Bryant at St. Vincent's Hospital, these symptoms were very noticeable upon the left side. I believe with Kraus that a more careful observation will reveal the existence of these symptoms on both sides in all cases of a crush of the lower part of the cervical and upper dorsal region of the spinal cord.

Thirdly. A careful study of the cases here collected furnishes additional evidence in regard to the localization of the motor functions of the spinal cord. They serve to confirm quite fully statements made by me in 1884 and again in 1888 in regard to the representation of the various muscles of the upper extremities in different segments of the cervical region.

In the articles alluded to, it will be found that every muscle is assigned a representation in more than one segment of the cord, and this fact has been accepted by Mills and by Kaiser. It is well known that the groups of cells in the anterior horns of the cord have a considerable extent vertically, so that some groups are long enough to reach through two, or even three segments, and but very few groups can be said to be situated in one segment only. But in as much as these groups of cells govern individual muscles, as we must believe in view of the facts known regarding anterior poliomyelitis, it is evidently a mistake to assign the representation of single muscles to single segments. I cannot, therefore, adopt the arrangement proposed by Thorburn and copied by Collins in which individual muscles are assigned to single segments.

If cases such as these here collected are carefully studied it will be found that a lesion limited to one segment rarely paralyzes any muscle completely. If, however, the lesion extends through two or more segments the paralysis is complete. Or if a lesion is a progressive one extending either upward or downward it will be

[3] Kraus. *Zeitschrift für Klin. Med.*, XVIII.

found that the muscles will be paralyzed in greater and greater degree as the lesion extends. These facts are only to be explained upon the theory that individual muscles have a considerable vertical representation in the cord, and this, as we have already seen, corresponds to the anatomy of the groups of cells. The table already published, therefore, presents the facts in an accurate manner.

I am quite prepared to admit that the vertical extent of individual groups of cells may vary in different individuals, thus in two cases carefully studied by Beevor and by Johnson the lesion though involving the sixth cervical segment caused no paralysis of the biceps, while on the other hand, in one of Herter's cases the biceps were decidedly involved by a lesion of the seventh segment. It is, therefore, probable that the representations of muscles are not always the same in different cords. The table, however, may be considered as fairly accurate because it combines a large number of facts derived from the study of very many cases. It is, perhaps, more accurate than the earlier tables. In recent cases care has been taken to examine the paralyzed muscles electrically and the existence of the reaction of the degeneration is certainly a positive proof that the group of cells governing the muscle has been destroyed.

In conclusion, I think it may be stated that the sensory and motor functions of the cervical region are now as well determined as those of the lumbo sacral region of the spinal cord.

Dr. LLOYD read a paper on

TRAUMATIC AFFECTIONS OF THE CERVICAL REGION OF THE SPINAL CORD SIMULATING SYRINGOMYELIA.

DISCUSSION.

Dr. WILLIAM OSLER, of Baltimore.—It is extremely interesting to note that these observations which enable us to localize lesions of the cord so accurately, come from the surgeon, the clinical physician, and the experimental physiologist. With reference to the ciliospinal centre, while we see myosis in spinal lesions, it is most frequent, perhaps, in aneurism pressing on the cer-

vical sympathetic, in which it is by no means uncommon to see preliminary dilatation and the subsequent development of myosis, which persists on the affected side. All the features of paralysis of the cervical sympathetic may not be present together; thus, we not infrequently see myosis without narrowing of the palpebral orifice, or retraction of the bulb. Dr. Osler then referred to a case of pressure on the cervical sympathetic due to a tumor secondary to epithelioma of the leg.

Dr. CHARLES K. MILLS.—This paper adds one more contribution of a valuable character to assist us in the work of exact diagnosis.

In speaking of segmental localization, Dr. Starr referred to the spinal representation of muscles. I believe that a better expression for the cord as well as for the brain is the "representation of movements." This helps us to simplify and to render exact our method of looking at this matter. The same is true with reference to sensory representation in the cord. It is a representation of areas in the skin,not of nerves. Indeed this has been indicated by all that has been said to-night.

This question of segmental spinal localization has, as we all know, been applied to the explanation of functional diseases, particularly by Head, and this is one of the most interesting matters in connection with the subject. I had hoped that Dr. Starr would have referred to this. Head takes herpes zoster to give him an objective method of demonstrating his points. He holds that we have in the eruption of this affection, segmental localization, and not as has been commonly taught, merely nerve representation; and he believes that many functional affections have areas of cutaneous hyperæsthesia which he attempts to show correspond very closely with the areas of skin in herpes, and that these represent the segments of the cord affected. This is carrying studies of this kind a step further in a very interesting direction. If the observations are correct, they will perhaps throw new light upon our views with reference to certain trau-matisms of the spinal cord, and will help us in our studies of hysteria.

With reference to Brown-Sequard paralysis, I do not think that the position cited by Dr. Lloyd with approval, can be sustained. The sensory decussation in the cord is cellular, rather than a mere fibrous decussation, as was formerly held. The view now is that the transmission of nerve impulses is by cells—by contact of cells and

thin processes—and not by continuity of fibres. This being the case, as I pointed out several years ago in an editorial article in the *Medical News*, the destruction of the spinal decussation by the lesions of syringomyelia, will account for the sensory phenomena.

The correct view according to the most recent investigations, would seem to be, that the decussations of the fibres which constitute the great sensory pathways for the forms of so-called common sensibility—touch, pain, temperature, etc.—take place in the cord and the oblongata in two ways: (1) by crossings in the spinal commissures at all levels of Golgi's commissural cells conveying the pathic and temperature impulses to Gowers's ascending antero-lateral tract; (2) by the crossing in block or mass of the tactile sensory tracts of the columns of Goll and Burdach, just above the cuneate and clavate nuclei at the so-called decussation of the fillet or upper sensory decussation. From this level upwards all the fasciculi for common sensibility pass cephalward together. According to this view, irregular sensory phenomena might arise in cases of peculiarly distributed unilateral lesions of the spinal cord—tactile and motor loss for instance, being shown on the side of the lesion, and impairment of the pain and temperature sense on the other side.

Dr. FRANCIS X. DERCUM.—With reference to the point in regard to the association of flacid palsies with exaggeration of the knee-jerk, we are reminded of the type of spinal syphilis which has been isolated by Erb, in which there is this flacid paralysis without a spastic condition and yet with an exaggerated knee-jerk. Unfortunately there are no autopsies to explain the seat of this lesion. However, in a number of these cases, there are also ataxic symptoms which would suggest that the postero-lateral columns are also involved. In other words, some of the cases suggest ataxic paraplegia. It may be that slight involvement of the postero-lateral columns in specific myelitis involving the pyramidal tracts accounts for the peculiar association of flacid paralysis with exaggerated knee-jerk.

Dr. G. BETTON MASSEY.—A case seen by me some five years ago offers corroboration of Dr. Starr's remarks in two instances. The case was that of a lady who had sustained a fracture of the sixth or seventh cervical vertebra, exactly which one was not determined, as a post-mortem was declined. There was complete

paralysis below the level of the shoulders, including the arms. She lived for ten months and died of intercurrent disease. Her condition five months after the accident was that of paralysis as mentioned; the arms were decidedly contractured, while below the level of the arms the paralysis was flacid. The reaction of degeneration was present throughout the paralyzed parts. There was also a hyperæsthetic area joining the paralyzed and healthy parts.

Dr. M. ALLEN STARR.—Brown Sequard paralysis presents one of the puzzles of neurology. It seemed to me in reading Head's paper, that possibly he might have found some explanation for the Brown-Sequard phenomena on the theory which he has developed so carefully of referred sensation. As I have pointed out, a patient who is totally anæsthetic below a certain line of the body feels a sensation produced in that anæsthetic area, not in the area where it is produced, but at a point in the hyperæsthetic zone above the zone of anæsthesia. It seems to me that it is not impossible that we may find some explanation of the Brown Sequard phenomena in such erroneous conscious reference of sensations which come from another source.

Dr. Lloyd alluded to Golgi's observations showing that most of the fibres pass up the same side of the cord. Yet some of Golgi's stained sections show that there are many fibres that do cross the cord in the posterior part of the commissural portion of the cord. In the last edition of Kölliker's book, the decussating fibres are pretty well demonstrated. I do not think that we can doubt the existence of decussating fibres and sensory decussation in the posterior portion of the cord. The facts are well summarized by Marie, in his last book, in which he describes a number of cords examined after long standing amputation of certain limbs. In these cords ascending degeneration was found not only on the side of the amputation, but also in the other side in the posterior columns of the cord. This is shown in his diagrams of degeneration in the tracts of the cord.

The statement that I made with regard to the cervical sympathetic on the authority of Kraus, was that in lesions of the cervical sympathetic nerve in the neck, there were not only disturbances of the pupil, narrowing of the palpebral fissure and retraction of the bulb, but also unilateral sweating and flushing of the face, and that when there was lesion of the cilio-spinal centre in

the cord, no flushing of the face nor sweating was present.

I do believe that movements are represented in the cortex, but it seems to me that the phenomena present in anterior poliomyelitis where individual muscles are picked out by the disease and atrophied, rather tends to the view that muscles are represented in the cord rather than movements. It does not seem to me that in anterior poliomyelitis you have a certain set of movements obliterated as you have in cortical lesions, and I would therefore make the distinction, that muscles are represented in the cord and movements in the brain.

With regard to Head's views; those with reference to herpes, I have been able to confirm, but those in regard to cutaneous hyperæsthesia from internal disease, I have been unable to confirm. Head holds, for instance, that in cancer of the stomach, there is pain not only in the region of the stomach, but also distinct cutaneous hyperæsthesia, which is limited to a definite area corresponding to the segment of the cord connected with the stomach. I have examined many cases of disease of the internal organs, but was not able to find a single instance in which there was the cutaneous hyperæsthesia described by Head.

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